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/s/ William J. Wiscott

Elective Induction of Labor in a Small Community Hospital— A Two-Edged Sword

NORMAN LEVIN, M.D.*

Practically all of the articles published on elective induction of labor have come from large teaching institutions. Most of the authors, such as Field,[1,2] D'Esopo,[3] Bishop,[4] Schaefer,[5] O'Leary,[6] and Taffeen,[7] favor this procedure when practiced in a prudent manner; but Eastman, Keettel,[8] Niswander and Patterson[9] maintain that there are a certain number of irreducible complications which are directly related to the procedure, and these hazards outweigh the advantages that are derived from the elective induction.

In Keettel's[8] series, the incidence of prematurity, prolonged latent period with intrapartum infection, and prolapse of the cord were increased. Niswander and Patterson[9] reported an 11.8% incidence of babies that were admitted to a special nursery in respiratory distress as a result of the induction when compared to 7% incidence of respiratory distress for those mothers who went into spontaneous labor. Wells,[10] in her report on induced versus spontaneous labor, states that delay in the onset of respiration for 6 minutes or longer occurred twice as often in the induced as in the spontaneous group.

Our purpose in this paper is to present the experience compiled on a group of electively induced patients by obstetricians practicing in a small community hospital where 60% of the obstetricians are board certified. The rest are board qualified except for one general practitioner who is allowed to do elective induction with consultation.

One might ask the question, "Why do elective induction of labor?" Advocates of this mode of delivery list the advantages from an anesthetic, nursing personnel, psychological viewpoint, etc., but I strongly suspect that the reason many elective inductions are performed is because the patient and obstetrician like the convenience of the procedure. The question to be answered by each hospital practicing elective induction is whether there is a price to be paid by the fetus and mother because of the elective induction.

Methods and Criteria

All of the cases were induced by the use of an oxytocic intravenous drip followed by amniotomy after the onset of regular uterine contractions, the rate of the flow of the drip being titrated against the character of the uterine contractions in an effort to obtain effective regular rhythmic uterine contractions.

It was necessary for the attending obstetrician or his representative to be in the hospital for the entire time of the induction while trained nurses' aides or nurses were in constant attendance at the patient's bedside. Fetal hearts were monitored only by the use of the stethoscope during this entire series.

* Lutheran Hospital, Baltimore Maryland

Selection of the patients for induction generally conformed to the prevailing standards of dilatation, effacement, and consistency for the negotiable cervix as well as the station and position of the presenting part, which should be a vertex. Despite the fact that only single vertex presentations were considered for elective induction in this study, 5 breeches and 3 sets of twins were inadvertently induced.

Results

This is a prospective study on 1,029 consecutive elective inductions performed from July 1, 1961 through April 1, 1965. During this time, there were 6,998 deliveries performed at the Lutheran Hospital of Maryland (14.7%). Each case was coded on a special study sheet which was completed by the obstetrical anesthesia department in order to be sure that the results would be reported in a detached and unbiased manner.

In the entire series of cases only 109 primigravidas were electively induced, in comparison to 920 multiparous patients (11%). Since the average obstetrician's practice usually comprises 25-30% primigravida patients, it is obvious that the primigravida patients were very carefully selected. Consultations by another qualified obstetrician were mandatory on all primigravidas that were to be electively induced. (Table I).

Table I Elective Induction Study

Dates of study: July 1, 1961 to April 1, 1965	
I. Total No. Deliveries during study period	6,998
II. Total Inductions	1,029
III. Primiparous patient	109
IV. Multiparous patient	920
Incidence of induction	14.7%
Mean length of labor:	
Primigravida patient	6 hrs. 32 min.
Multiparous patient	4 hrs. 4 min.

Fetal Complication

Table II reveals the fetal mortality and morbidity. There were 5 fetal deaths with 2 stillborn and 3 perinatal deaths for a perinatal mortality of .49%. Two fetal deaths were due to congenital anomalies incompatible with life, giving us a corrected perinatal mortality of .29%

There were 3 fetal deaths that were directly attributable to the procedure. One occurred in a 20-year-old, para 0-1-0-1, who was delivered of a 2150 Gm. infant after a four hour labor, the baby dying 36 hours after birth of subarachnoid hemorrhage. A second fetal death occurred in a 27-year-old, para 2-0-0-2, who delivered a stillborn after 5 hours and 45 minutes of labor. Autopsy studies on the second baby revealed only hypoxia as the cause of fetal death. The fetal heart, in this particular case, was not monitored closely enough to detect fetal distress prior to fetal death and thus prevent this catastrophe. A third preventable fetal death occurred as a result of the hypoxia caused by intrapartum rupture of the uterus.

Fifty-three babies had to be placed in an isolette following delivery because of cyanosis, grunting respirations, or retraction of the chest. During this same period, only 2.5% of babies over 2500 Gm. were admitted to an isolette in our

Table II Fetal Results & Complications of Study

Stillbirths	2
Neonatal Mortality	3
Perinatal Mortality (uncorrected)	.49%
Perinatal Mortality (corrected)	.29%
Respiratory Distress Syndrome	.53 or 5.2%
Bladder distention in baby (2500cc of fluids used during induction)	1
Shoulder Dystocia	1
Breech	5
Twins	3
Prematurity	11 or 1.1%

nursery for respiratory distress when the mothers went into spontaneous labor.

Of those babies admitted to the isolette for respiratory distress, the following factors were thought to be significant:

1. Prematurity: Four of 11 cases of premature infants required being placed in an isolette. In the 3 unrecognized sets of twins, 1 premature infant had respiratory distress.
2. Sedation given within one hour of delivery with superimposed general anesthesia. Forty of our 53 cases had general anesthesia.
3. Longer than average labor was noted in 15 of 53 cases.
4. Sixteen of 53 babies had a fetal heart rate above 160 on at least two occasions during labor.
5. The incidence of tetanic contractions was not carefully recorded; and thus, it cannot be stated what its role might have been in our respiratory problems.

There were 11 premature infants in this series of 1,029 elective inductions (1.1%). One of our neonatal mortalities occurred in a premature infant (9%), reemphasizing the hazard of prematurity in elective induction of labor. One baby in this series had a markedly overdistended bladder, necessitating catheterization. A review of the chart reveals that the patient received 2500 cc. of fluid and 30 units of oxytocin during her 8 hours of induction.

Maternal Complications

Table III lists the maternal complications.

There were 4 cesarean sections performed in this series, all occurring in multiparas. One was performed on the basis of a prolonged labor of 24 hours and a compound presentation with the presenting parts at –2 station and cervix 4 cm. dilated at the time of the cesarean section. Another was performed for a persistent brow presentation which may have been caused by early rupture of the membranes with the presenting part at –2 station. A third section was performed on the basis of obstructive breech labor due to a large congenital cyst of the fetal left kidney. This baby did not survive because of multiple congenital anomalies. The fourth cesarean section was performed because of fetal distress due to a ruptured uterus. Three of the 4 sections performed were probably preventable.

Table III Maternal Complications

1. Cesarean Section	4 or .39%
2. Prolapsed Cord	0
3. Hypogastric ligation	3
4. Ruptured uteri	2
5. Prolonged latent period	2 or .27%
6. Maternal mortality	0
7. Endometritis	2

No prolapsed cords occurred in this series of cases, despite the usual quoted incidence for all deliveries being around .5%. In view of the high incidence of amniorrhexis that has been practiced in this study, this result is remarkable. The routine of rupturing membranes after the onset of regular uterine contractions, with the presenting part fixed abdominally and fundal pressure holding the vertex up against the cervical os, is recommended.

There were 12 cases of post-partum hemorrhage (1.2%), which compares favorably with usual quoted incidence of post-partum hemorrhage of 2-10%. This was attributed to our continuing the oxytocic drip post-partum for at least 2 hours and thereby preventing uterine atony, one of the common causes of immediate post-partum hemorrhage.

A most serious complication was 2 ruptured uteri, giving an incidence much higher than the usual quoted figures of 1 per 1500-2000 cases. Both of these ruptures occurred in similar type patients, i.e., 30 years of age or over, Gravida V, and unfavorable cervix. In one, as mentioned previously, the diagnosis of rupture of the uterus was made intrapartum because of fetal distress. In the other, postpartum cervical inspection revealed a laceration which extended up into the lower uterine segment. Hypogastric ligation was performed in both instances, the uterus being left in situ in one patient after repair of the uterine rent, and a total hysterectomy was performed in the second case. These 2 cases emphasize that the elective use of oxytocic substances is not innocuous.

Two cases of prolonged latent periods occurred in which the induction did not come to a successful conclusion on the first attempt. In both instances, the oxytocic drip was restarted the following morning and labor successfully ended on the second day with a good fetal and maternal result.

There were 2 cases of endometritis in this study. Considering our average duration of labor, and the short length of time that the membranes were ruptured prior to delivery, this result is understandable.

Discussion

In order to justify elective inductions of labor, the risk to the mother and the fetus should be comparable to or less than that attained in a similar group of patients who go into spontaneous labor. This series comprising 1,029 cases represents every elective induction from July 1, 1961 to April 1, 1965, and, more importantly, was done in prospective fashion. The reported incidence of 14.7% falls well within the 20-25% which Buxton[11] maintains can be induced safely on an elective basis.

A salient feature of this study was the 5.2% incidence of fetal respiratory distress as compared with a 2.5% incidence associated with spontaneous labor. Niswander and Patterson[9] also reported an increase of respiratory distress syndrome in electively induced versus spontaneous onset of labor babies.

Since all of our patients had early amniotomies as a part of the induction, one wonders if central nervous system trauma was a cause of the respiratory difficulties. Vasicka and Hutchinson[12] state that after rupture of the membranes and the vertex becomes engaged, the pressure exerted on the fetal head may be ten times that of the actual fundal pressure and thereby can cause bradycardia due to compression of the skull even though the contractions are physiological. While the whole fetus is within the amniotic fluid, according to Pascal's law, the pressure exerted from the contracting myometrium may be transmitted undiminished equally upon the combined amniotic fluid and its contents, i.e., fetus, umbilical cord, and allantochorionic vessels.

The eventual mentality and coordination of these babies as they progress to adulthood remains unanswered. Niswander[13] et al., in a 4-year follow-up study of children delivered by elective induction, utilizing tests of functional development, were unable to demonstrate any increased risk of brain damage provided the fetus is mature at the time of delivery.

Our corrected perinatal mortality of .29% is good, but 3 perinatal deaths were directly attributable to the procedure. On the other hand, one cannot help but wonder if any babies in this series of cases were saved because of the

utilization of the elective induction of labor, for on rare occasions all of us have been faced with the dilemma of delivering a stillborn that had been seen 3 or 4 days previously and found to have no signs of fetal distress. Then, too, there is the inherent perinatal morbidity and mortality associated with the rapid unattended deliveries that occur on the way to the hospital.

When one causes a rupture of the intact uterus with the use of oxytocic substance, the selection of the patient was injudicious and the drug should not have been used in the first place. Rupture of the intact uterus, according to Pedowitz,[14] and Garnet,[15] carries a maternal mortality of between 3-14%. We noted two ruptured uteri in our series of 1,029 cases, which is in accord with Guttmacher's reported three ruptured uteri in 1,493 elective inductions. This incidence of rupture of the intact uterus should act as a deterrent to the elective induction of patients of high parity over 30 years of age, reemphasizing that oxytocic substances can be as dangerous as a "rattlesnake," and that the responsible physician should be capable of performing a rapid hysterectomy and hypogastric ligation under emergency conditions. In addition, the hospital in which he practices should have an adequate blood bank, good anesthesia, and facilities for immediate emergency surgery.

The incidence of prolonged latent period will definitely be reduced when IV oxytocin drip is added to amniorrhexis in induction of labor. Our incidence of prolonged latent period of .2% is definitely less than reported by Keettel *et al*[8] when amniotomy alone was utilized in the majority of cases to induce labor.

Except for Fields and Green,[1] most of the authors discourage elective induction in the nulliparous patient. Of our cases, 109 (10%) were primigravidas, revealing our hesitancy in inducing the patient with the untried pelvis. There were no stillborns, neonatal mortality, cesarean sections, ruptured uteri or prolonged latent periods encountered among the primigravidas in our series. Perhaps the required mandatory consultation was meaningful in causing the obstetrician to be more circumspect in his selection of the primiparous patient.

Summary

A series of 1,029 consecutive elective inductions of labor comprising an incidence of 14.7% of patients delivered in a small community hospital has been presented. Our method of induction has been dilute oxytocin drip followed by amniotomy after the onset of regular rhythmic uterine contractions. The incidence of respiratory distress in the induced babies was doubled when compared to those that went into spontaneous labor. Our uncorrected perinatal mortality was .49%, with a corrected perinatal mortality of .29%, but there were fetal deaths due to the procedure. The incidence of infection, prolapsed cord, post-partum hemorrhage, and prolonged labor was reduced, but the incidence of ruptured uteri was increased. No maternal deaths were noted in this group of patients. In this study, the carefully screened primigravida (10%-109 cases) had a lower complication rate than the multiparous patient.

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Effect of Intrathecally Administered Methotrexate† on the Central Nervous System of Man: Report of Three Cases

MICHAEL B. A. OLDSTONE, M.D.,* HOWARD WISOTZKEY, M.D.,** THOMAS LAU, M.D.***

THE ADMINISTRATION of the folic acid antagonist Methotrexate may produce striking, although temporary, remissions in leukemia.[1-3] Oral or parenteral administration at proper dosage levels yields sufficiently high blood levels to produce the desired effect. However, Methotrexate is poorly transported across the blood-brain-barrier with failure to reach therapeutic levels in cerebro-spinal fluid.[4-6] Thus, neoplastic cells that have entered the CNS may not be affected by tolerated effective plasma drug levels. Indeed, there are numerous clinical case reports of patients with acute leukemia who, while in hematological remission, have developed meningeal leukemia suggesting that this possibility is a real one. Meningeal leukemia has become a major therapeutic problem in these patients.

Welch suggested the use of intrathecal Methotrexate in order to minimize the opportunity for the selection of drug resistant cells, or the establishment of a nidus from which the systemic circulation could be invaded.[7] Similarly other investigators have employed the intrathecal route for the administration of Methotrexate in man.[4, 8, 9] With the increasing usage of intrathecal Methotrexate not only in the treatment of meningeal leukemia but also in the "prophylaxis" of meningeal leukemia[10] it is necessary to determine if intrathecal Methotrexate itself is cytoarchitecturally toxic to the nervous system of man.

Table I

Patient	*Age*	*Weight (kilos)*	*Total Dose of Intrathecal Methotrexate in Mgs.*	*Diagnosis*
E.E.	50	60.4	40	Acute lymphocytic leukemia
A.R.	36	54.5	28	Chronic granulocytic leukemia in acute stage
I.I.	29	60.3	50	Acute myelogenous leukemia

Table II Results of Lumbar Punctures

Patient	Opening Pressure (MM H_2O)	Cells (Per MM^3)	CSF Sugar/ Blood Sugar (MGM %)	Protein (MGM %)
E.E.*	350	90	28/100	82
	185	123	26/80	83
	190	19	5/88	99
	120	2	56/100	29
	95	6	87/140	30
A.R.*	120	170	Not done	Not done
	120	"normal"	Not done	Not done
I.I.*	230	150	60	45
	220	50	Not done	23
	"Increased"	15	60	28
	190	4	70	40

*—all cultures were negative

This study consisted of pathologic study of 3 cases of meningeal leukemia treated with intrathecal Methotrexate. Clinical data, type of leukemia and dosage of Methotrexate may be obtained from Tables I & II. Sections from lumbar, thoracic, cervical cord, medulla, pons, basal ganglia and cerebral cortex were examined. All sections were stained with hematoxylin and eosin and then reexamined with luxol fast blue, Nissl and Bodian techniques.

†Lederle's brand of amethopterin

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One case (E. E. NS-11509) will be presented in some detail.

E. E., University Hospital Case 299108

This 20 year old Negro male was admitted to the University Hospital, Baltimore, for evaluation of recurrent sore throat, anorexia, a 12 lb. weight loss and unexplained fever of 2 months duration.

Physical examination at this time was normal except for generalized lymphadenopathy, a right Bell's palsy and hepatosplenomegaly.

Initial blood and bone marrow cultures were sterile. Bone marrow examination was diagnostic of acute lymphocytic leukemia. Other abnormal laboratory determinations included: hemoglobin 7.7 grams; peripheral white blood cell count 100,000 composed almost entirely of mature lymphocytes; stool guaiac 3+; BUN 92 mgm.%; uric acid 13.8 mgm.%; alkaline phosphatase 7.8 units and thymol turbidity: 7.0. A widened mediastinal shadow was seen on chest x-ray.

The patient was treated with prednisone and 6-mercoptopurine. On this therapy he became afebrile, entered a clinical and hematologic remission and was discharged on maintenance dosages of 6-mercoptopurine and prednisone.

Five weeks following discharge, the patient was readmitted because of progressive lymphadenopathy. Again the lymph nodes were matted, rubbery, enlarged and occasionally tender. Bone marrow showed recurrence of acute lymphocytic leukemia.

Methotrexate was added to his medication but no remission was obtained. During the second month of hospitalization, the patient had a grand mal seizure. Lumbar puncture on the following day revealed an opening pressure of 350 mm. H_2O with 90 lymphocytes per mm.,^{3} a CSF sugar of 28 mg.% and protein of 82 mg.%. Cultures were negative. A diagnosis of meningeal leukemia was made and the patient given intrathecal Methotrexate. Systemic treatment during this time resulted in hematologic remission. The patient was symptom free till approximately 2 weeks following his initial seizure when he had a second seizure. Lumbar puncture again revealed findings compatible with meningeal leukemia. Intrathecal Methotrexate was reinstituted. However, the patient developed bronchopneumonia, progressive azotemia and alimentary tract bleeding and expired on the 69th hospital day.

General Autopsy Findings: The general autopsy showed gross leukemic involvement of lymph nodes, particularly the cervical and periaortic chains. Leukemic infiltrates were seen grossly in the liver and kidneys. These findings were confirmed histologically and involvement of the spleen was also demonstrated. The bone marrow was diagnostic of acute lymphocytic leukemia. The lungs showed severe, bilateral, organizing broncho-pneumonia. Examination of the gastrointestinal tract revealed stercoral ulceration as a cause of the gastrointestinal bleeding.

Neuropathologic Findings: Grossly the brain and spinal cord were normal. Histological findings with H & E revealed a few small cerebellar infiltrates composed of abnormal cells of the lymphocytic series. In one cortical section parenchymal perivascular neoplastic infiltrates were seen. (Figure 1) Nissl stained sections showed good preservation of cellular detail without evidence of chromatolysis or neuronal loss (Figure 2). There was no evidence of demyelinization in the sections subjected to the luxol fast blue techniques. (Figure 3). Bodian stains showed no evidence of axonal degeneration. Multiple sections from cauda equina, lumbar cord, thoracic cord, cervical cord, medulla, pons, mesencephalon, basal ganglia and right frontal cortex were unremarkable. There was no demyelinization, axon degeneration, chromatolysis or neuronal loss identified.

Discussion

Three patients with clinical signs of meningeal leukemia who had been treated with intrathecal Methotrexate were ex-

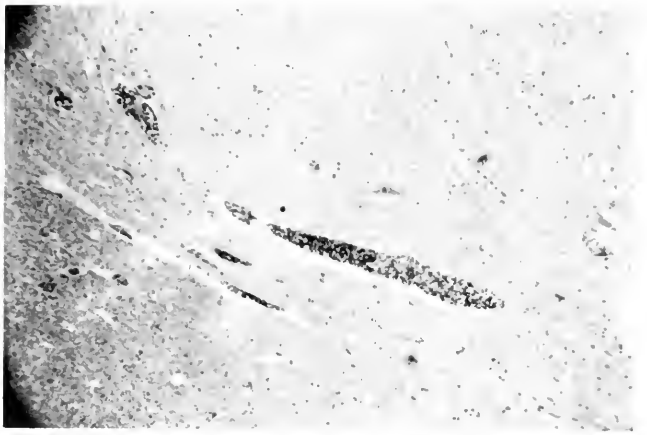

Fig. 1. Case E. E. NS-11509. Abnormal lymphocytic cells surround several cortical vessels. H & E X 100.

Fig 2. Case E. E. NS-11509. Histologically normal anterior horn neurons. Nissl X 430.

Fig. 3. Case E. E. NS-11509. Histologically normal lumbar spinal cord without evidence of demyelinization. Luxol fast blue X 10.

amined pathologically. One case has been presented in detail and all three cases are summarized in Tables I & II. All showed a decrease in the spinal fluid cell count following administration of the drug and none developed new neurologic signs after intrathecal chemotherapy. Pathologic study of these patients showed minimal evidence of meningeal infiltration at the time of autopsy. There was no histologic evidence of residual damage to nerve tissue which might be attributed either to pre-existing leukemic infiltration or to drug neurotoxicity. The presence of parenchymal cellular infiltrates seems to indicate that sufficient amounts of Methotrexate do not pass into the nervous parenchyma to effect neoplastic cells in that location.

Summary

Three cases of meningeal leukemia examined clinically and pathologically failed to show evidence of histologic change following intrathecal administration of Methotrexate. On the basis of this limited study, the administration of folic acid inhibitors by this route does not seem to produce detectable central nervous system damage.

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Effect of Repeated Partial Hepatectomies on Regeneration of Liver Autotransplants in the Rat*

ROGER LITTLE, AND PHILIP LITTMAN

The tremendous regenerative capacity of the liver in higher animals is well known. Earliest studies date back over 60 years.[15]

Recent work, mostly with rats, has involved removal of about 2/3 of the liver and then observing changes grossly, histologically, or biochemically for hours or days after partial hepatectomy until the liver grows back to its original size. It has been shown that DNA will increase within 24 hours and mitotic figures will reach a maximum number at about 48 hours.[8] The greatest rate of liver growth occurs within the first 4 days and by the second or third week the liver has regained its original size and weight.[10] One worker performed 5 partial hepatectomies at 5 to 7 week intervals and still was able to demonstrate a continuous capability for liver regeneration.[22]

The mechanism that signals the liver to regenerate has not been determined. The existence of some humoral factor differing from known hormones has been strongly considered. Early experiments involving parabiotic rats[2, 4, 26] and injections of serum or plasma[1, 11, 23, 27] suggested the possibility of a humoral substance in the serum of the partially hepatectomized rats capable of stimulating hepatocyte proliferation in a parabiotic or non-attached partner. However, others have not been able to reproduce these results.[5, 6, 12, 14, 16]

A fresh approach to this problem has been made with the use of liver autotransplants. If some blood-borne factor is involved in liver regeneration, then, an autotransplant should show the same or nearly the same changes as does the regenerating liver. Experiments have involved the establishment of autotransplants, subjecting animals to partial hepatectomy and then studying changes taking place in the autotransplant—grossly, histologically, or biochemically. Several workers claim to have evidence supporting the existence of a humoral factor in liver regeneration using this type of experiment.[13, 20, 25]

As was mentioned above, it has been shown that the liver is still capable of regeneration after multiple hepatectomies.[22] If a humoral factor does exist the parenchymal cell regeneration in the autotransplant as well as the residual liver should be stimulated by repeated hepatectomies. An autotransplant which is not stimulated in this manner should show only atrophic changes as described by other workers.[17, 19, 24]

Procedure: Two groups of Sprague-Dawley rats weighing 250-300 Gms. were used in this experiment. Group I was composed of rats with an autotransplant and no hepatectomy. These animals were kept alive for varying periods of time for study of a liver autotransplant without the influence of a possible humoral factor evoked by hepatectomy. Group II consisted of rats in which an autotransplant had been established and hepatectomy performed

* From the Department of Pathology, University of Maryland School of Medicine, Baltimore.

at regular intervals. All operations involved the use of a semi-sterile technique (Table I).

Table I

Group	Subgroup	Number of Hepatectomies	Duration of Transplants (wks.)	Number of Animals	
I	A	0	6	1	
	B	0	8	5	
	C	0	10	3	
	D	0	12	4	
	E	0	20	5	
				18	Total
II	A	2	6	3	
	B	3	8	4	
	C	4	10	1	
				8	Total

Using a method similar to that of Virolainen[25] an autotransplant was established by suturing the mesorchium to the liver and allowing three weeks for adequate adherence and vascularization. At the end of the three week interval a 1.0 cm^3 (approximate) piece of liver with the mesorchium well adhered was detached and placed in the lower abdominal cavity. In order to protect the newly established transplant from infection, Tetracyn,® 0.2 cc (5 mg) intraperitoneally was given one day prior to, the day of, and the day following surgery.

Group I animals were placed in subgroups and sacrificed periodically (Table I). The transplants were removed and paraffin sections were made for histological study.

Group II rats were divided into subgroups A, B, and C which received two, three, and four partial hepatectomies respectively (Table I). Group I animals served as controls and were not operated upon after the autotransplant had been established. Sham-operated controls were not felt to be needed because of adequately controlled experiments of other investigators who demonstrated no effect from sham operations.[13]

In Group II animals, hepatectomy was performed two weeks after establishment of the autotransplants. The first hepatectomy involved removal of 60-70% of the liver in all subgroups. A two week interval was maintained between partial hepatectomies in order to provide an adequate time for regeneration of the liver and autotransplant. Subsequent partial hepatectomies proved to be exceedingly more difficult due to adhesions and abscess formation; and smaller amounts of liver were removed at each subsequent partial hepatectomy. Estimated percent removed was 30-40% at second partial hepatectomy; 15-25% at third partial hepatectomy; and 10-20% at fourth partial hepatectomy. Subgroup A rats were sacrificed with controls two weeks after the second partial hepatectomy. Subgroups B and C were sacrificed with the controls for each subgroup two weeks after third and fourth partial hepatectomies respectively. Paraffin sections were made of all autotransplanted liver tissue.

Results

Grossly all autotransplants appeared as small pieces of brown tissue and were located in the lower abdominal cavity well away from the residual liver. Their sizes ranged from 2-5 $mm.^3$ There was no evidence of inflammation.

Autotransplants of Group II A showed no definite histological difference from those of Group I A. Both groups demonstrated active liver cells with prominent nuclei. Parenchymal cells were small with scanty cytoplasm and nuclei were close together. Basophilic material in the cytoplasm suggested actively growing cells but

mitotic figures were sparse. Some bile duct proliferation was seen along with retention of bile pigment simulating early biliary cirrhosis (Figure 1).

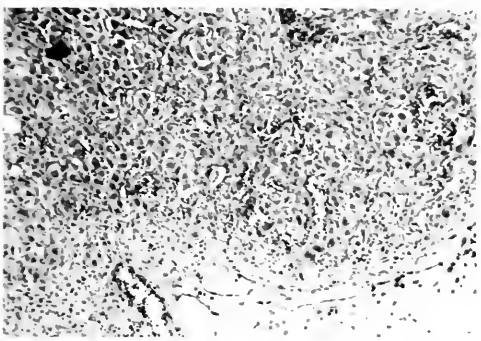

Figure 1. Six week autotransplant showing numerous liver parenchymal cells closely grouped with scant cytoplasm. Lobular structure cannot be delineated.

Group II B autotransplants were similar to those in Group I B. No evidence of active liver cell poliferation was seen. There were islands and some chords of parenchymal cells. Extensive scarring and bile duct proliferation were present which resembled severe biliary cirrhosis (Figure 2).

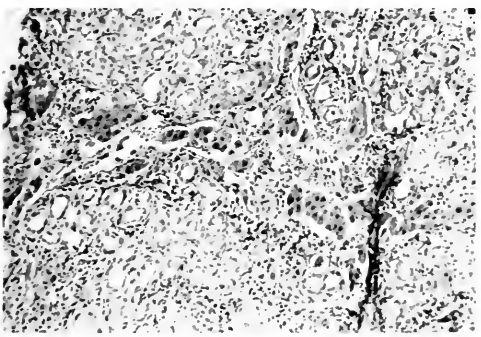

Figure 2. Eight week autotransplant demonstrating islands of liver parenchymal cells and extensive bile duct proliferation.

Group II C and Group I C autotransplants were similar histologically with extensive bile duct proliferation and scarring. Islands and occasional chords of liver cells persisted (Figure 3).

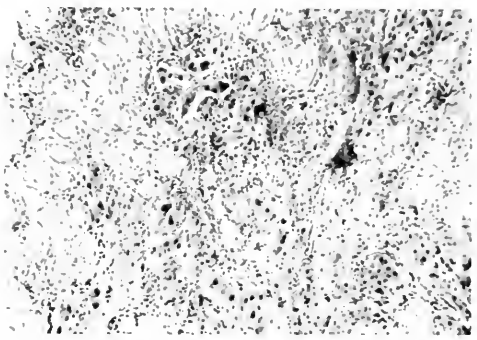

Figure 3. Ten week autotransplant showing bile duct proliferation and scattered islands and chords of liver parenchymal cells.

Group I D and Group I E autotransplants both demonstrated a marked proliferation of bile ducts with extensive fibrosis and scarring. Group I D still showed islands and some chords of liver cells. Nucleoli were prominent and there was some vesiculation of cytoplasm (Figure 4). Group I E had few surviving parenchymal cells with many large cystic bile ducts (Figure 5).

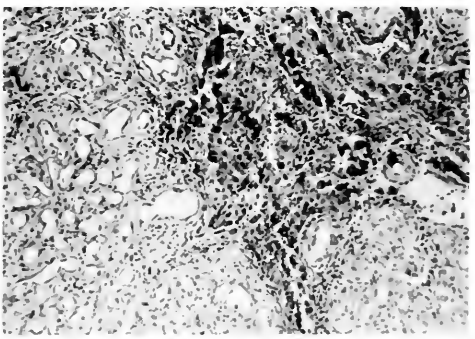

Figure 4. Twelve week autotransplant showing extensive bile duct proliferation and some groups of liver parenchymal cells with prominent nuclei.

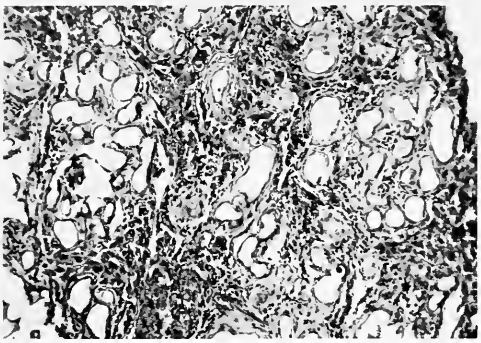

Figure 5. Twenty week autotransplant with many large cystic bile ducts and a few surviving liver parenchymal cells.

Discussion: The histological findings in the autotransplants were consistent with those of other investigators[24, 17] who showed few surviving parenchymal cells, fibrosis and great proliferation of bile ducts.

The lack of histological difference between our autotransplants and controls suggests that the stimulus for liver regeneration is only initiating cellular proliferation in the remaining portion of the partially hepatectomized liver. Numerous workers have proposed that the stimulus for liver regeneration is brought about by some hormonal substance with broader effects.

One might glean certain information about the humoral substance from work on liver regeneration *per se*. Bucher *et al* showed that after a 10% hepatectomy there is evidence of cellular proliferation at the periphery of the lobules only; and with larger partial hepatectomies cellular proliferation proceeds toward the center of the lobules.[3] This suggests that if a humoral factor exists it is affecting more liver cells as its concentration increases due to the amount of liver removed and may be acting along a concentration gradient. It may be deduced that a hormonal substance would act on those cells coming into contact with it first and in highest concentrations, i.e. those cells nearest incoming blood vessels. Some recent work by Siegel *et al* supports this concept.[20]

In our work the first and second hepatectomies should have produced adequate amounts of humoral substance because of the sufficient amount of liver removed for this effect.[3] One might expect that even the small amount of potential humoral substance produced as a result of the third or fourth hepatectomy might be enough to affect some change in the hepatocytes closest to good blood supply.

We examined surviving hepatic cells and cell groups carefully especially around blood vessels and saw no evidence of stimulated hepatocyte proliferation when comparing experimental and control autotransplants.

One might argue that hepatocytes in autotransplants cease in their ability to respond to the humoral substance due to age, poor vasculature or poor bile duct drainage. However, the hepatocytes appear normal histologically. It has been shown that they will respond to epinephrine after one year.[18] Our observations along with others show evidence of linear arrangements[17, 24] suggesting some slight inherent ability for regeneration. Furthermore, some proliferative response has been claimed due to one partial hepatectomy in autotransplants without special techniques for maintaining portal or bile duct integrity.[25]

Several workers give evidence of a regenerative response in the autotransplant due to partial hepatectomy. When the autotransplant has an intact bile duct and portal circulation, its regenerative response is identical to that of the partially hepatectomized liver; when only the bile duct remains intact, it responds less well.[8] And when neither the bile duct or portal vasculature is intact, it responds least well,[25] if at all.

Certain investigators have proposed that the liver regenerates because of physiologic demands[7] or because of a drop in plasma proteins.[6] Recent work with exchange transfusions between animals that have had partial hepatectomies and animals that have intact livers has shown that the blood from animals with intact livers has a slight inhibiting effect on the timing of the appearance of regenerative changes in liver. Blood from animals with partial hepatectomy has no effect at all on animals with intact or regenerating livers.[9] Thus, perhaps normal blood exchanged into an animal with a partial hepatectomy maintains for a short time some necessary balance.

One can explain the results of those who have suggested, because of the proliferative response of autotransplant hepatocytes, that some humoral factor exists which is responsible for liver regeneration. The autotransplant, depending upon how closely it resembles normal liver, has the capacity to react in the same way that the remainder of the hepatectomized liver does to some metabolic or physiologic demand brought about by partial hepatectomy. The autotransplants established by others were simply acting as ectopic liver.

In our experiments, as the autotransplant underwent atrophic changes the surviving parenchymal cells did not demonstrate any regenerative response to multiple partial hepatectomies. It can be postulated that our surviving hepatocytes for some unknown reason were not reacting to the humoral factor. This was discussed earlier in this paper. Furthermore, there has never been any real demonstration of the humoral factor itself. Several workers including ourselves have failed to find any evidence supporting its existence. Those that have may have been showing some other type of mechanism responsible for liver regeneration, i.e., demand or metabolic imbalance.

The debate concerning the existence of a humoral factor responsible for liver regeneration will continue. Our work adds more skepticism. Definitive proof awaits the demonstration of the humoral factor.

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Book Reviews

Diabetes for Diabetics. By George F. Schmitt, M.D., F.A.C.P. 237 pp. (illustrated), 1965. The Diabetes Press of America, Inc., 30 S. E. 8th Street, Miami, Florida.

This manual presents a fresh approach to the education of the diabetic. Chapters describing the disease itself, and discussing the discovery, action and manufacture of insulin and oral antidiabetic drugs allow understanding of physiology, pathology, and pharmacology not usually available to laymen. Appropriate questions following each chapter will stimulate the studious reader.

The patient is guided to an understanding of the physician by sections explaining the meaning of the physical examination, goals of treatment, the responsibilities of physician and patient, and the excellent glossary. Self-care hygiene instructions should help the reader to avoid preventable complications. Orientation of the diabetic within family and community is enhanced by sections relating diabetes to marriage, pregnancy, employment, insurance, and taxation.

The book is attractive and sturdily bound. The numerous illustrations, all in color, of urine testing, insulin administration, and food portions should attract the juvenile reader. The dietary exchange lists are unusually complete. Summer camps, and societies and associations for diabetics are listed with addresses.

Doctor Schmitt, an alumnus of the School of Medicine of the University of Maryland, has combined a complete instruction manual for the diabetic patient and a compact reference text for the layman into a single volume. *Diabetes for Diabetics* is a book for serious study by the diabetic who desires as complete an understanding of his disease and its treatment as a layman can achieve.

CHARLES E. SHAW, M.D.

Mongolism. Edited by F. E. W. Wolstenholme, F.R.C.P., and Ruth Porter, M.R.C.P. 95 pp., 1967. Little, Brown and Company, Boston. (Ciba Foundation Study Group No. 25)

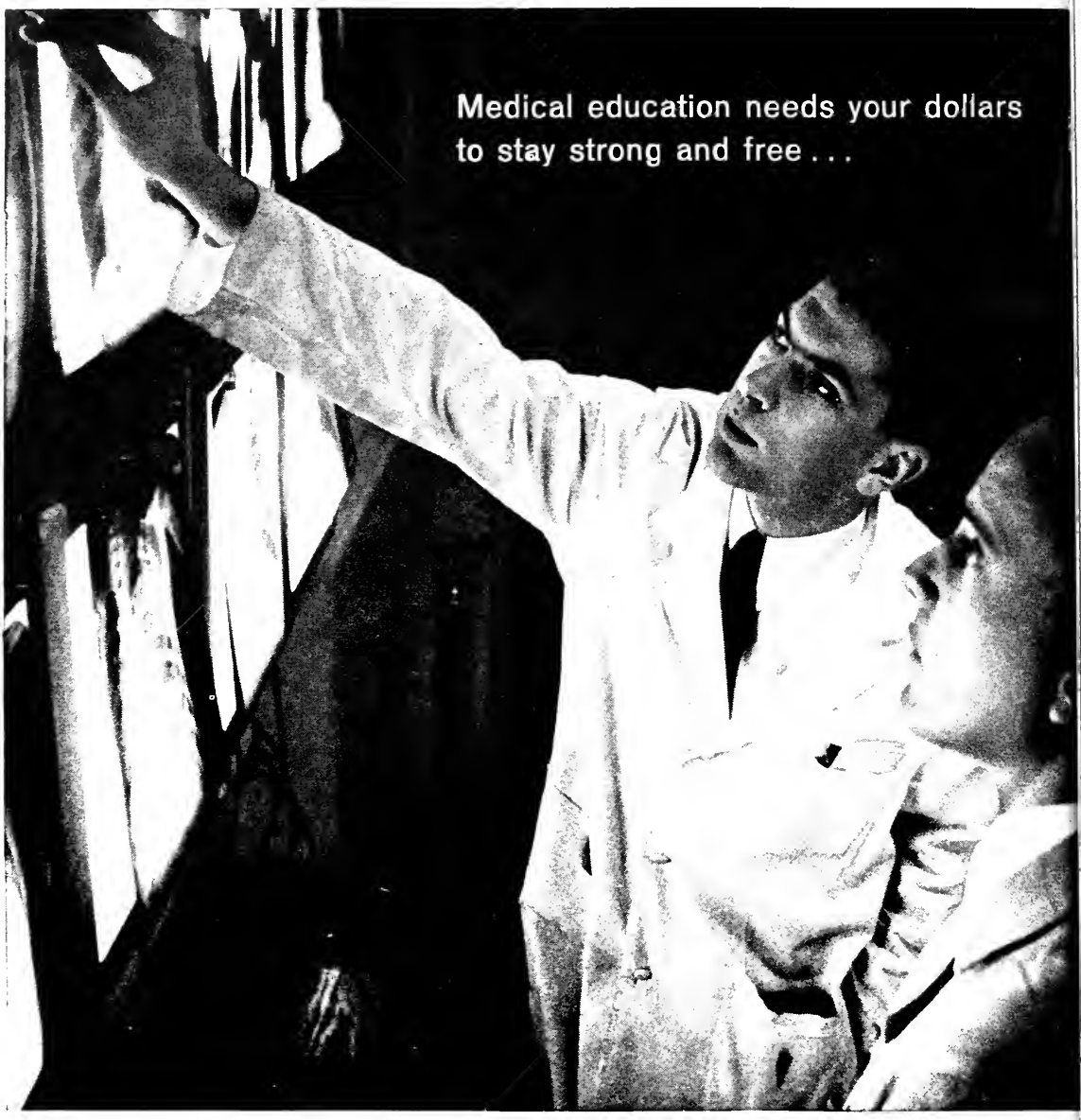
This book represents an attempt to describe mongolism as a disease associated with chromosomal abnormalities. The opening remarks by Lord Brain provide an excellent historical background of the subject and the remainder of the book contains several papers relating to the various clinical and biochemical aspects of the disease.

Such questions as the relationship of maternal age to incidence and the statistical significance of consanguinity are discussed in light of substantial data obtained through statistical studies.

The last few papers deal with DNA synthesis and replication patterns in mongoloids—an area which has only recently been explored. In a rather interesting fashion, the book describes and attempts to explain the various abnormalities in cell production as a result of trisomy at chromosome No. 21. Arguments to the contrary are proposed in the last paper dealing with granulocyte kinetics.

Certainly the book would be most informative to those physicians interested in mongolism. The subject matter is easily understood and the book can be read in one evening.

JOHN ECKHOLDT, M.D.



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MEDICAL SCHOOL SECTION

Dean's LETTER

Dear Members of the Medical Alumni, Students and Friends of the Medical School:

In 1961 this Medical School requested the Maryland State Planning Commission to make a study of Maryland's needs for medical education and medical research. The study was made and the report published in 1962. This report recommended that the University of Maryland Medical School expand its entering class to 155 students by 1970. The report recognized that this could not be done without the State providing the resources needed for this increased enrollment of medical students.

The report was referred to the University of Maryland for its reaction to the proposal. President Elkins appointed a Committee chairmanned by Dr. R. Lee Hornbake with membership from both the Medical School and University. After a careful study, the Hornbake Committee agreed that the expansion could be made if the State provided the clinical facilities, an addition to the Basic Science building and an operating budget commensurate with the student body enrolled.

The Medical School and University have moved progressively in the accomplishment of this expansion. Support for the clinical resources has been obtained, the curriculum has been revised, the faculty staffing initiated toward required levels and we have requested planning and construction money for the addition to the Basic Science building. We regret to inform you that the Governor has not approved the request for planning and construction of the Basic Science addition. It is possible that this still may be granted by the Legislature, but if it is not approved this year, we will not be able to expand the entering class to 155 in 1970.

Sincerely,

WILLIAM S. STONE, M.D.
Dean

January 1968

ALUMNI ASSOCIATION SECTION

President's Letter

Fellow Medical Alumni:

Much has occurred since my last letter. The Alumni Association and the Postgraduate Committee, under the able direction of Dr. Ephraim Lizansky, produced a very successful gathering of alumni at Amityville. This was made possible by the enthusiasm of Dr. Benjamin Stein and his many alumni friends of the New York area. I know that the faculty members who participated in the program found this meeting an enjoyable and stimulating one, as did the Amityville Alumni group.

A luncheon was held in Washington, D. C., on November 22, 1967, at the Statler Hilton Hotel. Arrangements for this were made by Dr. William Holbrook. Dr. Elkins, President of the University of Maryland, discussed the future plans for medical education, and Dr. William Stone, the Dean, spoke about the changing face of the medical school campus and its educational trends.

It is with regret that we learn that Dr. Stone has announced his planned retirement as Dean of the School of Medicine, effective the end of this academic year. He has been a dynamic force in establishing a fulltime faculty, modernizing the curriculum, building and broadening a research program, and in the construction of many physical improvements. I know he would like nothing better than to see continued change for the improvement of our school, and I think it is incumbent upon each and every member of this organization to see that Dr. Stone's successor is the most dynamic and capable physician available to continue the heritage of this Medical School, to produce physicians to care for the people of Maryland or wherever they may practice. Such an important decision must weigh heavily upon Dr. Elkins and the Board of Regents, and your support is essential.

December 21, 1967

Very sincerely,

John O. Sharrett M.D.

JOHN O. SHARRETT, M.D.

Complete Program for 1968 Alumni Day

Hospital Societies and Medical Alumni to Meet June 6 and 7

Medical Alumni Day Activities

Thursday, June 6, 1968

8:30-10:00 A.M. REGISTRATION and Coffee—*Davidge Hall*

9:00-12:00 A.M. SCIENTIFIC SESSIONS

University of Maryland Hospital Medical Association—*Gordon Wilson Hall*

The Douglass Obstetrical and Gynecological Society of the University of Maryland—*Psychiatric Institute, first floor, room 1-621*

The Bradley Pediatric Society of the University of Maryland—*Anatomical Hall, third floor, Davidge Hall*

University of Maryland Surgical Society—*Auditorium, Psychiatric Institute, room 1704*

12:10-1:00 P.M. ANNUAL ALUMNI BUSINESS MEETING AND PRESENTATION OF HONOR AWARD AND GOLD KEY TO THOMAS B. TURNER, M.D., Class of 1925—*Chemical Hall*

1:00-2:00 P.M. LUNCHEON—*Gymnasium, fifth floor, Psychiatric Institute*
(Complimentary tickets available at registration desk.)

2:00-4:00 P.M. SCIENTIFIC SESSIONS
(Choose from among those listed below.)

6:30 P.M. COCKTAIL PARTY for Fifty-year Graduates—*Florentine Room, Lord Baltimore Hotel*

7:00 P.M. BANQUET—*Ballroom, Lord Baltimore Hotel*

Introduction of Class of 1918

Recognition of Class of 1968

Speaker—Watch for further announcement

Dancing to follow, courtesy of Medical Alumni Association—Orchestra

All-day parking will be provided for alumni at the lot at Baltimore and Arch Streets, with the entrance from Arch Street only. The charge will be 45¢.

Medical Alumni Day, Thursday, June 6, 1968

Dr. Thomas B. Turner
To Receive
1968 Alumni
Honor Award

Dr. Thomas Bourne Turner, Dean of the Johns Hopkins University School of Medicine, has been nominated recipient of the 1968 Alumni Honor Award and Gold Key. The award, to be presented at ceremonies on June 6, will mark the completion of 11 years' service as the eighth dean of the Johns Hopkins School of Medicine and the 43rd anniversary of his graduation from the School of Medicine of the University of Maryland. Dr. John O. Sharrett, president of the Medical Alumni Association, will present the award in recognition of Dr. Turner's important contributions to medicine and for his outstanding service to mankind.

In 1917 Dr. Turner left his home in Prince Frederick, Maryland, to attend St. John's College in Annapolis, from which he was graduated in 1921. As a student on Greene Street, he came under the influence of the late Gordon Wilson, Walter Baetjer, Harry M. Stein, Hugh R. Spencer and Drs. Shipley, Dobbin and Bergland. Shortly after his graduation, he met the late Dr. Maurice C. Pincoffs, and became one of his first residents at Mercy Hospital in Baltimore. In 1927 he was appointed Jacques Loeb Fellow in Medicine at the Johns Hopkins University School of Medicine, where he ultimately joined the Department of Medicine.

Shortly after his appointment, he became associated with Dr. Alan M. Chesney, who was at the time investigating a number of spirochetal diseases

under the auspices of the Rockefeller Foundation. Dr. Turner soon found himself in the Caribbean studying Yaws and other similar conditions, an interest which he continues to maintain. Having completed his assignment, he returned in the mid-thirties to positions with the Department of Medicine and the Public Health Administration. During this time, he completed more than two dozen studies, relating primarily to spirochetal disease. In 1939 he was named Professor of Microbiology, an appointment he still retains.

With the advent of World War II, he became head of the venereal disease program of the Army Surgeon General's Office, ultimately serving as military government Health Officer in Europe and North Africa.

Returning to his duties in the Department of Microbiology, Dr. Turner reactivated his interest in treponemal disease, publishing a number of important papers on the subject. Many of these were cooperative studies, either basic or clinical in orientation, and related primarily to the public health aspects of such disorders. He is coauthor, with Dr. D. H. Hollander, of a monograph entitled *The Biology of Treponematoses.*

When the Johns Hopkins Medical Institutions began their expansion programs during the early 1950s, the University selected him to lead and to help plan the enormous development of research and clinical facilities which has characterized the noteworthy progress made during the past decade.

An experienced scientist and educator, he accepted the challenge, completing the task with a distinction which will remain as a monument to his tenure. As the eighth academic dean of the School of Medicine, Dr. Turner has developed and effectively instituted a conjoined collegiate and medical school curriculum and provided for the orderly expansion of research and clinical facilities, while maintaining the traditional image and balance of the institution.

Dr. Turner has been honored by many learned and scientific societies. He has served as a member and as president of the Executive Council of the Association of American Medical Colleges, and has written a number of articles as well as a monograph, published in 1963, on the fundamentals of medical education. He has been honored with membership in the Association of American Physicians, the Harvey Society, the American Society for Clinical Investigation and a number of high ranking professional societies relating to public health, microbiology and social hygiene.

For several years he has been a member of the Board of Visitors and Governors of St. John's College. In 1966 the degree of Doctor of Science, Honoris Causa, was conferred upon him by the University of Maryland.

On July 1, Dr. Turner will leave his post as dean of the Johns Hopkins to return to his work in clinical medicine and in original research for the Department of Microbiology. He has accepted the challenge of writing the fourth volume of the history of the Johns Hopkins Hospital and of its School of Medicine, a task begun by his old friend and preceptor, the late Alan M. Chesney. Thus, on the one hundred and sixty-first anniversary of the founding of the School of Medicine and on the forty-third anniversary of his graduation, it is appropriate that Thomas B. Turner, distinguished alumnus, scientist, investigator, educator, counselor and friend, be awarded the highest honor and recognition offered by fellow alumni, the Honor Award and Gold Key of the Medical Alumni Association.

SCIENTIFIC PROGRAMS

University of Maryland Hospital Medical Association

Thursday, June 6, 1968

SCIENTIFIC SESSION

Gordon Wilson Hall

Moderator—C. CONOVER TALBOT, M.D.

9:30-9:50 A.M. "Studies of Renal Tubular Function in a Patient with the Idiopathic, Adult Fanconi Syndrome"
WILLIAM F. FALLS, JR., M.D., *Staff Physician, Veterans Administration Hospital, Richmond, Virginia*

9:55-10:15 A.M. "Detection of Occult Bone Metastases of Breast Cancer at the Time of Radical Mastectomy by Photoscanning with Strontium-85"
N. DAVID CHARKES, M.D., *Department of Nuclear Medicine, Temple University Hospital, Philadelphia, Pennsylvania*

10:20-10:50 A.M. "Management of Renal Failure"
GLENN D. LUBASH, M.D., *Associate Professor of Medicine, Head of Division of Hypertensive—Renal Disease, University of Maryland School of Medicine*

10:55-11:25 A.M. "Physiology of Heat Stroke"
SHELDON E. GRIESMAN, M.D., *Associate Professor of Medicine, University of Maryland School of Medicine*

11:30 A.M. BUSINESS MEETING

12:30 P.M. COCKTAILS AND LUNCHEON—*Caesar's Forum, Holiday Inn, Howard and Lombard Streets, Baltimore, Maryland*

The Douglass Obstetrical and Gynecological Society

SCIENTIFIC SESSION

Psychiatric Institute, first floor, Room 1-621

9:00 A.M. "Study of Retinal Vessels in Patients Taking Oral Contraceptives"
A. MEISELS, M.D., *Assistant Professor of Ophthalmology,* and EDMUND B. MIDDLETON, M.D., *Associate Professor of Obstetrics and Gynecology*

"Induction of Ovulation with Humegon"
BOBBY A. RIMER, M.D., *Assistant Professor of Obstetrics and Gynecology*

"Experimental Viralizing Tumor in Human Ovarian Tissue"
ISADORE G. ANCES, M.D., *Instructor in Obstetrics and Gynecology*

"Treatment of Carcinoma of the Cervix"
UMBERTO VILLASANTA, M.D., *Assistant Professor of Obstetrics and Gynecology*

The Bradley Pediatric Society

BUSINESS AND SCIENTIFIC SESSION

Anatomical Hall, third floor, Davidge Hall

Chairman—SAMUEL S. GLICK, M.D., *President*

2:00-2:30 P.M. Welcome
KARL H. WEAVER, M.D., *Acting Head, Department of Pediatrics*

2:30-3:10 P.M. "Legislation, and the Effect on Pediatric Practice"
SAMUEL P. BESSMAN, M.D., *Professor of Pediatric Research and Professor of Biochemistry*

3:10-3:20 P.M. Discussion and Questions

3:20-4:00 P.M. "The Operation of the Community Pediatric Center"
RAY HEPNER, M.D., *Director, Community Pediatric Center, Professor in Pediatrics*

4:00-4:10 P.M. Questions

University of Maryland Surgical Society

SCIENTIFIC SESSION

Auditorium, Psychiatric Institute, Room 1704

Moderator—EDWIN R. JENNINGS, M.D.

9:00-9:10 A.M. Welcome
ROBERT W. BUXTON, M.D. and ERWIN R. JENNINGS, M.D.

9:10-9:40 A.M. "Combat Surgery in Vietnam"
LEONARD W. GLASS, M.D.

9:45-10:00 A.M. "Intravenous Nutritional Support of the Non-Alimenting Patient"
BRUCE H. MACPHERSON, M.D.

10:00-10:15 A.M. "Treatment of Hydrocephalus: Direct Attack on the Obstruction"
ROBERT M. N. CROSBY, M.D. and RONALD L. PAUL, M.D.

10:15-10:30 A.M. "The Uses, Techniques and Results of Autogenous Tibial Cortical Bone Plates"
T. HUGH MORGAN, M.D.

10:30-10:45 A.M. Coffee Break

10:45-11:00 A.M. "Blood Lactate as an Index of Severity in Various Forms of Clinical Shock"
VLADIMIR VITEK, PH.D., ELWOOD LABROSSE, M.D., PH.D. and R. ADAMS COWLEY, M.D.

11:00-11:15 A.M. "Clinical Uses of Refractive Index"
DAVID R. BOYD, M.D. and ARLIE R. MANSBERGER, JR., M.D.

11:15-11:30 A.M. "Electrical Injuries to the Skull"
C. PARKE SCARBOROUGH, M.D. and WILLIAM H. MOSBERG, M.D.

Thursday Afternoon, June 6, 1968

SCIENTIFIC SESSION

Auditorium, Psychiatric Institute, Room 1704

Moderator—JAMES G. ARNOLD, M.D.

2:00-2:15 P.M. "Carotid Artery Endarterectomy"
JOSEPH S. MCLAUGHLIN, M.D., and C. THOMAS FLOTTE, M.D.

2:15-2:30 P.M. "The Significance of Extra-Luminal Aortic Perfusion Following Acute Thoracic Dissection"
NORMAN H. BAKER, M.D.

2:30-2:45 P.M. "Use of Dextran in Treatment of Atherosclerotic Occlusive Disease"
C. THOMAS FLOTTE, M.D.

2:45-3:00 P.M. Coffee Break

3:00-3:15 P.M. "The Acute Symptomatic Aneurysm—A Surgical Emergency"
THEODORE DODENHOFF, M.D. and EVERARD F. COX, M.D.

3:15-3:30 P.M. "Replacement of Infected Arterial Prostheses with Inverted Jejunal Autografts"
PHILIP J. FERRIS, M.D. and CSABA MAGASSY, M.D.

3:30-3:45 P.M. "Basilar Artery Insufficiency Due to Atlanto-Occipital Instability"
HERBERT S. BELL, M.D.

3:45-4:00 P.M. "Bleeding in the Surgical Patient"
SAFUH ATTAR, M.D., JOSEPH S. MCLAUGHLIN, M.D. and R. ADAMS COWLEY, M.D.

FRIDAY, JUNE 7, 1968

SCIENTIFIC SESSION

Auditorium, Psychiatric Institute, Room 1704

Moderator—WILLIAM B. LONG, M.D.

9:00-9:15 A.M. "The Transversalis Fascia: A Practical Analysis of an Enigma"
ROSS Z. PIERPONT, M.D.

9:15-9:30 A.M. "Plastic Repair of Traumatic Skull Defects"
HANS R. WILHELMSEN, M.D., C. PARKE SCARBOROUGH, M.D., PAUL D. MEYER, M.D. and CHARLES M. HENDERSON, M.D.

9:30-9:45 A.M. "Current Methods in the Treatment of Finger Tip Injuries"
JAMES W. STRICKLAND, M.D. and DAVID L. DINGMAN, M.D.

9:45-10:00 A.M. "The Radical Mastectomy Incision"
DAVID B. GRAY, M.D.

10:00-10:15 A.M. Coffee Break

10:15-10:30 A.M. "A Simple Treatment of Chronic Lower Extremity Stasis Ulcers"
MICHAEL B. FLYNN, M.D. and C. THOMAS FLOTTE, M.D.

10:30-10:45 A.M. "The Water-Retining Lipid(s) of Eschar—Their Presence and Potential Usefulness"
CARL JELENKO, III, M.D.

10:45-11:00 A.M. "Prostatism and Infected Urachal Cyst—A Differential Diagnosis"
LOUIS C. BRESCHI, M.D.

11:00-11:15 A.M. "Organ Preservation—Studies Related to Storage of Donor Kidneys"
WILLIAM S. KISER, M.D.

11:15-11:30 A.M. "The Salivary Gland Scintiscan in Inflammatory Disease"
MARGARET M. FLETCHER, M.D. and JOSEPH B. WORKMAN, M.D.

11:30-12:00 A.M. BUSINESS MEETING OF THE SOCIETY

6:00 P.M. BUSSES LEAVE HOTEL FOR GIBSON ISLAND

7:00 P.M. COCKTAILS AND DINNER—*Gibson Island Club*

LADIES' ACTIVITIES

Wednesday, June 5, 1968

8:30-11:00 P.M. All ladies are invited to attend the RECEPTION AND COCKTAIL PARTY to be held at the *Hospitality Center in the Caswell Room of the Lord Baltimore Hotel*

Thursday, June 6, 1968

9:00-9:45 A.M. HOSPITALITY—*Embassy Room, Lord Baltimore Hotel*—Coffee and Sweet Rolls

9:45 A.M. BUS TOUR—Leaves promptly from *Hanover Street Entrance, Lord Baltimore Hotel*

Baltimore Museum of Art, Mr. Jack Stephens, Curator of Education (Bus will leave Museum at 11:45 A.M.)

Historic Hampton House, Mrs. David McPherson, Curator

1:30 P.M. LUNCHEON—*Hampton House Tea Room* (Bus will leave for Hotel at 3:00 P.M.)

Price $5.00. Please place reservations by May 31, 1968.

Mrs. Howard B. Mays, *Chairman,* Mrs. Walter E. Karfgin, *Co-chairman,* Mrs. Everett S. Diggs, Mrs. Lewis P. Gundry, Mrs. John O. Sharrett, Mrs. Wilfred H. Townshend, Jr. and Mrs. Gibson J. Wells

Class NOTES

Your achievements, fellow alumnus, are of interest to your classmates. They constitute a reward to the faculty, are a challenge to the younger physicians, and are an item of prestige for the University. Please cooperate with us by forwarding news of yourself or any alumnus to the BULLETIN. *Thank you.*

CLASS OF 1897

Dr. William Richard Arthur, now nearly 92, was born on the old Daniel Boone Trail in Southeastern Kentucky on a section of land that was awarded to his great-grandfather by Thomas Jefferson for services rendered the nation during the Revolutionary War.

Dr. Arthur, whose picture is reproduced herewith, was asked to reminisce about his long life and of his many years in general practice. This, he stated, could best be expressed by way of his professed hobby, that of writing poetry. Dr. Arthur's poem is reproduced herewith. Dr. Arthur's permanent retirement address is 14201 N.W. 17th Avenue, Miami, Florida.

A PATIENT WHOM I CANNOT FORGET

I once had a patient too ill to get well,
Whose pastor had preached about the flames of hell!
His vivid depictions filled her with dismay
For his hell sounded worse than that of Dante.
There's no hope for me, moaned the poor old dame
As she thought of her torture in a red hot flame.

Convinced of her fate, she had but one thought
Hell's gaping for me, I'm as good as caught.

When she sent for me she explained her plight
But failed to tell me her reason for fright.
If mortal sin, 'twas a secret well kept,
Though she raved about hell except when she slept.
But the cancer she had could not be excised, and she never slept but when narcotized.
Her constant plea was to hold death at bay,
That she'd stay out of hell just one more day.

She could not conceive death was her best friend
That only it could bring her woes to an end.
She lingered for weeks in that hell of her own,
With each unnarcotized pang a sobbing moan.
To be kept alive, and away from hell's flame . . .
And her niece accused me of being to blame
For the drawn out hours of Aunt's agony,
With the sordid thought of augmented fee.

DR. WILLIAM RICHARD ARTHUR, 1897 P&S

CLASS OF 1939

Dr. Thomas S. Sexton, Vice President and Chief Medical Director of the Massachusetts Mutual Life Insurance Company, has been elected Vice President of the Association of Life Insurance Medical Directors of America.

A member of the Association since 1947, Dr. Sexton has served on the Association's executive committee and also holds membership in the Insurance Medical Group of New England and the medical section of the American Life Convention. He also is medical director's representative of the Life Insurance Medical Research Fund.

ALUMNI NEWS REPORT

THE BULLETIN:

I would like to report the following:_____

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SUGGESTIONS FOR NEWS ITEMS

American Board Certification
Change of Address
Change of Office
Residency Appointment
Research Completed
News of Another Alumnus
Academic Appointment
Interesting Historic Photographs

Name_____

Address_____

_____

Class_____

Send to

Dr. John A. Wagner, Editor
Bulletin—School of Medicine
University of Maryland
31 S. Greene St.
Baltimore, Md. 21201

A native of Sistersville, West Virginia, Dr. Sexton has been associated with Massachusetts Mutual since 1947. He was elected vice president and chief medical director in 1962 and a member of the company's board of directors in 1965. He serves also as chairman of the board of trustees of the Belchertown State School and vice-chairman of the Springfield area Community Health Study. He is also a corporator of the American International College and Springfield Institution for Savings.

CLASS OF 1951

Dr. David M. Kipnis, Professor of Medicine and Director of the Washington University Clinical Research Center, participated as a member of the faculty in an intensive post-graduate course entitled "Diabetes in Review" held on January 24, 25 and 26 at the Sheraton-Cleveland Hotel in Cleveland, Ohio.

The program under the auspices of the American Diabetes Association, in cooperation with the Case Western Reserve University School of Medicine and the Cleveland Clinic, featured many authorities on the subject of diabetes, its cause, its pathologic effects and treatment.

Dr. Kipnis, known for his many contributions to the field of endocrinology, is the recent recipient of the endocrine society's Ernst Oppenheimer Memorial Award sponsored by the Ciba Pharmaceutical Company. This prize honors accomplishments in endocrinology by a person under 41 years of age.

Dr. Kipnis also won the Lilly award of the American Diabetes Association for his work on intra-cellular transport in its relation to phosphorylation of glucose.

CLASS OF 1958

Dr. Robert J. Robl, Lieutenant Commander, Medical Corps (USN), is currently serving as chief of radiology at the U. S. Naval Hospital in Oakland, California.

CLASS OF 1960

Dr. I. William Grossman has been named associate pathologist at the Sinai Hospital of Baltimore.

A native of Baltimore, Dr. Grossman is an alumnus of both the School of Medicine and the School of Pharmacy.

Following his graduation, he trained in pathology at the Mount Sinai Hospital in New York and subsequently served as a fellow at the same institution. In addition to his duties at the Sinai Hospital, Dr. Grossman will serve as an assistant professor of pathology at the School of Medicine at the University of Maryland and as an instructor at the Johns Hopkins University School of Medicine.

A former faculty member of the College of Physicians and Surgeons, Columbia University, Dr. Grossman also served as captain in the Army Reserve Medical Corps, doing research in the development and pathology of subcellular organelles, using electron microscopy. Dr. Grossman is a diplomate of the American Board of Pathology in both anatomic and clinical pathology.

CLASS OF 1965

Dr. Timothy K. Gray has received a Southern Medical Association grant in aid relative to his training in internal medicine. Dr. Gray currently serves as an assistant resident at the University of Maryland Hospital.

Alumni Medical Reunion 1968

Dr. Wilford H. Townsend reported that each of the Hospital Societies (medical, gynecological, pediatric and surgery) has agreed to join the Medical Alumni Association at a joint meeting to be held June 5, 6 and 7, 1968. Dr. Conover C. Talbot, President of the Hospital Medical Association, will come from Chicago for the meeting with various other society heads and the Alumni Association representatives.

BULLETIN *School of Medicine*
University of Maryland

VOLUME 53 APRIL, 1968 NUMBER 2

TABLE OF CONTENTS

Primary Ovarian Pregnancy:

Report Of A Case & Review of Literature

IN-JOO PARK, M.D., and I. A. SIEGEL, M.D.

TRUE OVARIAN PREGNANCY is a rare complication of pregnancy. While many authorities doubt the possibility of a primary implantation of the ovum in any tissue other than that of Mullarian origin, an increasing number of primary ovarian pregnancies are being reported. In 1941 Curtis[8] stated that among 100 cases recorded, approximately 60 cases were authentic. Baden and Heins[4] believed that well over 100 cases had been reported prior to March, 1951. Haselhorst, in 1953 (quoted by Procope[15]), stated that 240 such cases had been recorded. Babrow and Winkelstein[2] found 155 cases as of 1956.

The incidence of ovarian pregnancy among total deliveries has been reported as 1 in 24,453[4] and 1 in 5,900.[5] In our hospital it was 1 in 19,440 deliveries. It is generally believed that the incidence of ovarian pregnancy to total intrauterine pregnancies is 1 in 25,000 to 40,000.[9, 12, 13, 21] Its occurrence in ectopic pregnancies is reported as 1 in 117;[4] 1 in 251;[5] 1 in 348;[3] 1 in 232;[15] 1 in 339;[6] 1 in 110.[12] At Franklin Square Hospital it was 1 in 120. Thus the incidence in ectopic pregnancy is somewhere between 1 in 120 to 348.

Case History

A 20-year-old white female, para 0-0-0-0, was admitted to this hospital on August 16, 1965, with complaints of sudden onset of right lower abdominal pain with fainting. The patient had been married for the past eight months. Her last menstrual period started on July 6, 1965, and its duration was four days as usual. *At the time of admission she was ten days past due.* The pain was sharp and colicky in nature, not associated with any fever, nausea or vomiting. The pain occasionally radiated to the opposite side of lower abdomen and to the epigastrium. She felt dizzy and fainted frequently on the day of her admission. The patient also had dysuria but no frequency. Her bowels were loose with the onset of symptoms.

Past and Family History: Noncontributory.

M.H.: 13 x 28-30 x 4-5—regular and normal.

Physical Examination: General Condition: Markedly pale looking, well-developed female, not in acute distress. B.P. 110/70. Pulse 100/min. weak. Respirations 20/min. regular.

Head: ENT. Negative.

Neck: No palpable node. Thyroid not enlarged.

Chest: Heart and Lungs—normal. Breast—no mass, no discharge.

Abdomen: Soft. Scaphoid type. No mass palpated, no rigidity noted. There was marked tenderness on the right lower quadrant and some in the left lower quadrant. Rebound tenderness was positive on the right side.

Pelvic Examination: External genitalia—normal with female escutcheon. Marital introitus—vaginal wall well supported. No glands were felt. Cervix—no erosion, no bleeding and closed. It was tender on motion. Corpus—anteverted, movable and slightly enlarged. Soft and tender. Adnexae—no palpable mass. Markedly tender on right side. Cul-de-Sac bulging.

Laboratory Data: Ht. 28. WBC 8,500 Neuto-

From Franklin Square Hospital, Baltimore, Maryland.

philes 82, lymphocytes 13, monocytes 5. Urinalysis—negative. Pregnancy test—negative.

With the impression of ectopic pregnancy, culdocenthesis was performed, yielding about 20 ml. of nonclotting blood. The patient underwent exploratory laparotomy immediately following culdocenthesis and blood transfusion of 1000 ml. was started.

Operative Finding: A low midline incision was made, the peritoneum was opened and found to be filled with 500 cc. consisting of both old blood and free fresh blood. After aspiration of blood, the uterus and tubes were examined. The uterus was found to be slightly enlarged and soft. Both tubes were intact and perfectly normal in appearance including the fimbriated ends. There was no evidence of bleeding from the tubes. Left ovary was inspected and found to be normal. The right ovary was slightly enlarged and at the tip of the right ovary there was a small ruptured cyst containing a blood clot. The size of cyst was about 2 x 1 x 1 cm. The right ovary was connected to the uterus by ovarian ligament. The diagnosis was of right hemorrhage corpus luteum with rupture, and a wedge resection of this area was done. The patient recovered uneventfully and was discharged a week later.

Pathology Report: Gross: The specimen consisted of an 8 x 7 x 5 cm. blood clot and a 2.5 x 1.5 cm. portion of ovary which on the surface had a 1 cm. ruptured hemorrhagic cyst.

Microscopic Finding: Unfortunately it was impossible to reconstruct the original shape of the ruptured sac, but ovarian tissue occupied the wall of the entire gestational sac except where it was ruptured. There was no connection to any other tissue from this area of rupture.

The sections of the ovary contained a depressed hemorrhagic cystic space on the serosal surface. This space was in part lined by decidua with a few fragments of trophoblastic tissue embedded in fibrin on the surface of the decidua. The central portion of the cyst was filled with hemorrhage. The remaining portion of the ovary contained several dilated follicles and a few corpora albicans. The blood clot contained numerous chorionic villi lined with intact cytotrophoblasts and knots of syncytiotrophoblasts. The stroma of the villi was quite loose. Fragments of an early embryo were found in the amnionic sac. (See Figures 1 to 4.)

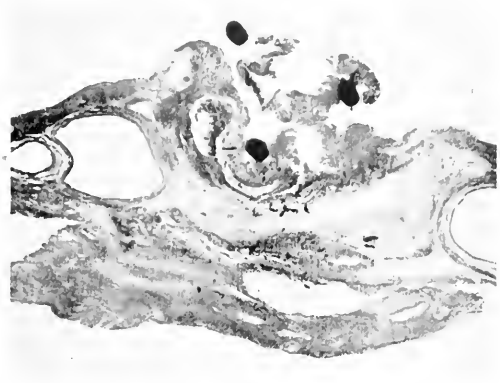

Figure 1. 4X. Section from resected ovary, showing depressed hemorrhagic area of implantation.

Figure 2. 4X. Section from blood clot which contains trophoblast and fragments of early embryo in amniotic sac.

Figure 3. 120X. High power view of embryo and amniotic sac.

Etiology

The etiology of ovarian pregnancy is not definitely agreed upon. Several hypotheses have been developed to explain the mechanism of this phenomenon. Leopold (quoted by Curtis in 1910[8]) stated that "all primary ovarian pregnancies result solely from the fertilization of the ovum before it escapes from the Graffian follicle." He believed that fertilization occurs in the ovary when the ovum is prevented from escaping from the follicle. This idea is disputed by some on the basis that the corpus luteum is essential for the developing ovum and that the growing trophoblastic tissue would thus destroy the corpus luteum of the ovary. Rock[4, 16] disagrees with Leopold, since the presence of blood in the vicinity of the ruptured follicle makes the sperm incapable of dispersing hyaluronic acid (present in the granulosa and pelucidal layers of the ovum) and the hyaluronidase would be neutralized by the antihyaluronidase contained in the blood.

Others[5, 7, 14] suggest that ectopic endometrosis may be the site of ovum implantation. Gerin-Lajoie[10] states that "the great frequency of the presence of endometrium in the ovary favors the nidation of the egg within the cortex and that fecundation of the ovum at the cortex of the ovary at the site of follicular rupture is generally the rule." Despite this, Baden and Heins[4] in 97 cases of ovarian pregnancy found only two cases associated with ovarian endometriosis. Procope[15] in his series failed to demonstrate a single case of endometriosis.

Ashley[1] investigated the hypothesis of ovarian pregnancy as being of parthenogenetic origin. In 12 cases he found male nuclear sex in 7 and female nuclear sex in 5, thus concluding that ovarian pregnancy is not parthenogenetic and that

Figure 4. 120X. High power view of amniotic sac and chorionic villi.

fertilization must precede ovarian implantation of a zygote.

Recently a number of authors[1, 4, 6, 8, 9] suggest that fertilization of the ovum may take place after follicular expulsion, either in the tube or peritoneal cavity with the fertilized ovum subsequently implanting on the cortex of the ovary or in the corpus luteum of the ovary.

Dowling[9] postulates two possible mechanisms of ovarian pregnancy:

1. Cortical Implantation—implantation into the cortex of the ovary following fertilization of ovum in tube or peritoneal cavity.
2. Interstitial Implantation—rupture of follicle with failure of ovum to be extruded, followed by fertilization of the ovum while still in the ruptured follicle.

Classification of Ovarian Pregnancy

Baden & Heins[4] classify ovarian pregnancy as:

1. *Primary Ovarian Pregnancy*

   a. The ovarian tissue forms a complete intact layer around the fetal tissue. Thus in the case of a ruptured ovarian pregnancy, the site of rupture must be through a

previous intact layer of ovarian tissue and other adherent extraneous tissue or organs will not alter this classification.

b. Intra-follicular. The fertilized ovum is implanted and grows in the Graffian follicle.

c. Extra-follicular. The fertilized ovum is implanted and develops in ovarian tissue other than in the Graffian follicle. This type would include juxto-follicular, interstitial, cortical and superficial implantations

2. *Combined Ovarian Pregnancy.* This group would include all ovarian pregnancies in which the ovary comprises at least a portion of the implanted fertilized ovum but does not form the entire wall, i.e., the remaining wall could be made up of other organs or tissue.

In 1918, Spiegelberg[19] set forth the following criteria for diagnosis of an ovarian pregnancy:

1. That the tube, including the fimbra ovarica, must be intact and separate from the ovary.
2. That the gestational sac must definitely occupy the normal position of the ovary.
3. That the sac must be connected with the uterus by the ovarian ligament.
4. That unquestioned ovarian tissue must be demonstrated in the walls of the sac.

It is interesting to note that decidual reaction is rarely found in ovarian pregnancy,[18] although it is commonly found in tubal pregnancy. It is believed that decidual reaction may be identified in early intact ovarian pregnancy, whereas in late ovarian pregnancy the true nature of this tissue may become obscured.[9]

Discussion

According to Dowling,[9] the first recognition of primary ovarian pregnancy is attributed to Saint-Maurice of Perigord, France, in 1682, but Gilford[11] believes the first instance on record goes to Bernitz and Goupil.

The diagnosis of ovarian pregnancy usually cannot be made prior to a complete pathological examination of tissue removed.[17] The symptomatology and physical findings are usually those of ectopic pregnancy.

The mean age of its occurrence[4, 15] is from 27 to 30 years. Ovarian pregnancy has a greater chance of going to term than a tubal pregnancy because the involved ovary is expendable. However, superficial cortical implantation is more easily ruptured in the early stages of gestation. Thus two-thirds terminated in the first trimester, one-fourth in the second trimester and one-fourth in the third trimester of pregnancy. There is a high incidence of stillbirths in viable ovarian pregnancies as well as grossly malformed fetuses.

Many early cases of ovarian pregnancy may be missed because of the diagnosis of ruptured corpus luteum and cyst or hemorrhagic follicular cyst. Only a careful pathological study of the tissue removed may result in the true diagnosis.[17]

The treatment of ovarian pregnancy usually is the same as for early ectopic pregnancy, i.e., remove the total product of conception and for a more advanced abdominal pregnancy, i.e., remove only the fetus and leave the attached placenta in place.

Summary

1. A case of primary ovarian pregnancy which fulfills Spiegelberg's criteria

was reported. It was an extra-follicular type with rupture.

2. Etiology of an ovarian pregnancy is not well understood.

3. It is rare to find a decidual reaction in ovarian pregnancy.

4. It would be simpler to include in the term "primary ovarian pregnancy" all cases where the fertilized ovum is implanted in or on the surface of the ovary.

5. The primary ovarian pregnancy may be subdivided into intra-follicular, stroma, cortical, and superficial types according to the site of growth of the fertilized ovum, regardless where fecundation has taken place.

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DEPARTMENT
OF
OBSTETRICS & GYNECOLOGY
UNIVERSITY OF MARYLAND
SCHOOL OF MEDICINE

Annual Report

Summary of Admissions, Discharges

and

Perinatal Mortality

University Hospital

January 1, 1966 through December 31, 1966

UNIVERSITY HOSPITAL

Baltimore, Maryland 21201

Obstetrical Report for the University Hospital

For Period January 1, 1966 through December 31, 1966

I. SUMMARY

	White	Non-White	Total
Total Discharges	742	1828	2570
Total Deliveries	702	1716	2418
Multiple Pregnancies			
Twins (No. of sets)	4	18	22
By Cesarean section	0	3	3
Triplets (No. of sets)	0	1	1
By Cesarean section	0	0	0
Total Adult Deaths	0	3	3
Rates per 1000 live births	0.00	1.78	1.26
Total Live Births	692	1688	2380
Total Fetal Deaths	10	28	38
Rate per 1000 total births	14.25	16.32	15.72
Total Neonatal Deaths	5	35	40
Rate per 1000 total births	7.12	20.40	16.54
Total Perinatal Mortality	15	63	78
Rate per 1000 total births	21.37	36.71	32.26
Perinatal Mortality (1000 grams & over)	12	41	53
Rate per 1000 total births	17.24	24.29	22.23

II. TOTAL DISCHARGES BY TYPE OF DELIVERY

	White	Non-White	Total
Abortion*, completion of	0	2	2
Abortion, spontaneous	2	7	9
Abortion, therapeutic	0	0	0
Ectopic pregnancy, early	0	0	0
Ectopic pregnancy, late	0	0	0
Full Term, spontaneous delivery	271	810	1081
Full Term, operative delivery	356	601	957
Premature†, spontaneous delivery	32	143	175
Premature, operative delivery	37	134	171
Immature‡, spontaneous delivery	5	16	21
Immature, operative delivery	1	12	13
Postpartum admission	3	14	17
Discharged undelivered	35	89	124
Not pregnant	0	0	0
Died undelivered	0	0	0
Total Discharges	742	1828	2570
Percentage	28.9	71.1	100.0

* An abortion is any fetus or infant weighing between 0-499 gm.

† A premature is any fetus or infant weighing between 1000-2499 gm.

‡ An immature is any fetus or infant weighing between 500-999 gm.

III. TOTAL DISCHARGES BY REASON FOR ADMISSION

	White	Non-White	Total
True labor	507	1319	1826
Suspected labor	19	47	66
Elective induction	27	4	31
Indicated induction	7	4	11
Postpartum admission	4	14	18
Ectopic pregnancy	0	0	0
Elective section	12	36	48
Abortion, threatened	0	0	0
Abortion, completion of	2	10	12
Abortion, therapeutic	0	0	0
Obstetrical disease	153	357	510
Medical disease	10	36	46
Surgical disease	1	1	2
Mole and Chorio-carcinoma	0	0	0
Not pregnant	0	0	0
Special study	0	0	0
Total	742	1828	2570

IV. SERVICE STATUS

Race	Private		Ward		Total	
	No.	%	No.	%	No.	%
White	488	65.8	1785	97.6	2273	88.4
Non-White	254	34.2	43	2.4	297	11.6
Total	742	100.0	1828	100.0	2570	100.0

V.—A AGE AND PARITY—TOTAL PATIENTS DELIVERED

White Discharges

AGE	PARITY												Total	Perinatal Mortality	
	0	1	2	3	4	5	6	7	8	9	10 & Over	Unknown		No.	%
Under 15	4	0	0	0	0	0	0	0	0	0	0	0	4	0(0)	0.0
15–19	106	37	9	2	0	0	0	0	0	0	0	0	154	4(1)	2.6
20–24	83	79	46	17	10	9	0	0	0	0	0	0	244	6(2)	2.5
25–29	16	37	38	30	27	14	4	0	0	0	1	0	167	3(0)	1.8
30–34	9	16	22	13	10	11	4	1	4	0	1	0	91	0(0)	0.0
35–39	1	2	4	3	4	3	4	3	3	1	0	0	28	1(0)	3.6
40–44	0	1	2	2	2	2	2	1	0	0	1	0	13	1(0)	7.7
45–49	0	0	0	0	0	0	0	1	0	0	0	0	1	0(0)	0.0
50 and over	0	0	0	0	0	0	0	0	0	0	0	0	0	0(0)	0.0
Total	219	172	121	67	53	39	14	6	7	1	3	0	702	15(3)	2.1
Perinatal Mortality No.	6(2)	5	0	1	2	0	0	1	0	0	0	0	15(3)		
Per Cent	2.7	2.9	0.0	1.5	3.8	0.0	0.0	16.7	0.0	0.0	0.0	0.0	2.1		

The numbers in ( ) indicate immature deaths.

V.—B AGE AND PARITY—TOTAL PATIENTS DELIVERED

Non-White Discharges

AGE	PARITY												Total	Perinatal Mortality	
	0	1	2	3	4	5	6	7	8	9	10 & Over	Unknown		No.	%
Under 15	15	1	0	0	0	0	0	0	0	0	0	0	16	1(1)	6.3
15–19	373	143	45	11	2	0	0	0	0	0	0	0	574	22(7)	3.8
20–24	113	114	151	82	35	25	4	1	1	1	0	0	527	14(6)	2.7
25–29	19	36	45	47	36	35	27	13	8	3	1	0	270	14(5)	5.2
30–34	7	12	15	21	33	26	25	26	10	12	9	0	196	8(2)	4.1
35–39	3	6	5	6	16	14	14	6	10	6	12	0	98	2(0)	2.0
40–44	0	0	4	2	5	3	2	5	0	1	8	0	30	2(1)	6.7
45–49	1	0	1	0	0	0	1	0	1	1	0	0	5	0(0)	0.0
50 and over	0	0	0	0	0	0	0	0	0	0	0	0	0	0(0)	0.0
Total	531	312	266	169	127	103	73	51	30	24	30	0	1716	63(22)	3.7
Perinatal Mortality															
No.	18(6)	10(4)	12(4)	6(1)	2(1)	3(0)	6(2)	2(0)	2(2)	0(0)	2(2)	0(0)	63(22)		
Per Cent	3.4	3.2	4.5	3.6	1.6	2.9	8.2	3.9	6.7	0.0	6.7	0.0	3.7		

VI.—A PRENATAL CARE—TOTAL PATIENTS DELIVERED

Number of Prenatal Visits	White		Non-White		Total		Perinatal Mortality	
	No.	%	No.	%	No.	%	No.	%
0	33	4.7	46	2.7	79	3.3	4(3)	5.1
1–3	90	12.8	320	18.6	410	17.0	35(12)	8.5
4–6	138	19.7	423	24.7	561	23.2	12(1)	2.1
7–9	170	24.2	504	29.4	674	27.9	3(1)	0.4
10–12	98	14.0	212	12.4	310	12.8	5(0)	1.6
13 or more	36	5.1	76	4.4	112	4.6	5(0)	4.5
Elsewhere	53	7.5	43	2.5	96	4.0	0(0)	0.0
Unknown	84	12.0	92	5.4	176	7.3	14(8)	8.0
Total	702	100.0	1716	100.0	2418	100.0	78(25)	3.2

VI.—B TIME OF FIRST VISIT

	White		Non-White		Total		Perinatal Mortality	
	No.	%	No.	%	No.	%	No.	%
13 weeks or earlier	34	4.8	113	6.6	147	6.1	3(1)	2.0
14–27 weeks	307	43.7	979	57.1	1286	53.2	33(8)	2.6
28 weeks or later	145	20.7	397	23.1	542	22.4	16(2)	3.0
Unknown	130	18.5	138	8.0	268	11.1	22(11)	8.2
Total	616	87.7	1627	94.8	2243	92.8	74(22)	3.3

VII. PRESENTATIONS—TOTAL INFANTS

Presentation	White		Non-White		Total		Perinatal Mortality	
	No.	%	No.	%	No.	%	No.	%
Vertex	657	93.6	1632	95.1	2289	94.7	50(11)	2.2
Breech*	32	4.6	58	3.4	90	3.7	22(12)	24.2
Face	3	0.4	4	0.2	7	0.3	1(1)	14.3
Brow	2	0.3	0	0.0	2	0.1	0(0)	0.0
Compound	0	0.0	2	0.1	2	0.1	0(0)	0.0
Transverse	3	0.4	11	0.6	14	0.6	4(1)	28.6
Unknown	5	0.7	9	0.5	14	0.6	1(0)	7.1
Total	702	100.0	1716	100.0	2418	100.0	78(25)	3.2
Twins	8	1.1	36	2.1	44	1.8	8(2)	18.2
Triplets	0	0.0	3	0.2	3	0.1	1(0)	33.3
Weight	*Breech Perinatal Mortality							
500–999 gm.	0	0.0	13	22.4	13	14.4	12	92.3
1000–1499 gm.	1	3.1	5	8.6	6	6.7	6	100.0
1500–1999 gm.	3	9.4	4	6.9	7	7.8	2	28.6
2000–2499 gm.	6	18.8	10	17.2	16	17.8	2	12.5
2500 gm. & over	22	68.8	26	44.8	48	53.3	0	0.0
Total	32	100.0	58	100.0	90	100.0	22	24.4

Mortality 1000 grams and over—13%.

VIII. METHOD OF DELIVERY—TOTAL INFANTS

	White		Non-White		Total		Perinatal Mortality	
	No.	%	No.	%	No.	%	No.	%
A. Vaginal Deliveries	665	94.7	1589	92.6	2254	93.2	69(25)	3.1
1. Total forceps deliveries	325	46.3	565	32.9	890	36.8	5(0)	0.6
Low forceps, elective	284	40.5	468	27.3	752	31.1	5(0)	0.7
Low forceps, indicated	2	0.3	2	0.1	4	0.2	0(0)	0.0
Mid forceps, elective	37	5.3	89	5.2	126	5.2	0(0)	0.0
Mid forceps, indicated	2	0.3	2	0.1	4	0.2	0(0)	0.0
High forceps	0	0.0	0	0.0	0	0.0	0(0)	0.0
Vacuum extractor, elective	0	0.0	0	0.0	0	0.0	0(0)	0.0
Vacuum extractor, indicated	0	0.0	0	0.0	0	0.0	0(0)	0.0
Failed forceps/extractor	2	0.3	1	0.1	3	0.1	0(0)	0.0
2. Breech	28	4.0	49	2.9	77	3.2	22(12)	28.6
Spontaneous	0	0.0	2	0.1	2	0.1	2(2)	100.0
Assisted	5	0.7	8	0.5	13	0.5	0(0)	0.0
Extraction	23	3.3	39	2.3	62	2.6	20(10)	32.3
Decomposition & Extraction	0	0.0	0	0.0	0	0.0	0(0)	0.0
3. Other operations	4	0.6	8	0.5	12	0.5	0(0)	0.0
Version & extraction (single)	1	0.1	0	0.0	1	0.0	0(0)	0.0
Version and extraction (second twin)	0	0.0	0	0.0	0	0.0	0(0)	0.0
Manual rotation, head only	2	0.3	8	0.5	10	0.4	0(0)	0.0
Rotation of shoulders	1	0.1	0	0.0	1	0.0	0(0)	0.0
Destructive operations	0	0.0	0	0.0	0	0.0	0(0)	0.0
4. Spontaneous	308	43.9	967	56.4	1275	52.7	42(13)	3.3
B. Abdominal deliveries	37	5.3	127	7.4	164	6.8	9(0)	5.5
1. Cesarean section	36	5.1	127	7.4	163	6.7	8(0)	4.9
2. Rupture of uterus	1	0.1	0	0.0	1	0.0	1(0)	100.0
3. Advaneed ectopic pregnaney	0	0.0	0	0.0	0	0.0	0(0)	0.0
GRAND TOTAL	702	100.0	1716	100.0	2418	100.0	78(25)	3.2

IX. ANCILLARY OPERATIVE PROCEDURES FOR LABOR AND DELIVERY

	White		Non-White		Total		Perinatal Mortality	
	No.	%	No.	%	No.	%	No.	%
A. Induction of labor								
Oxytocic	71	10.1	81	4.7	152	6.3	3(1)	2.0
Rupture of membranes	1	0.1	0	0.0	1	0.0	0(0)	0.0
Rupture of membranes and oxytocic	0	0.0	0	0.0	0	0.0	0(0)	0.0
Stripping of membranes	1	0.1	2	0.1	3	0.1	0(0)	0.0
Stripping of membranes and oxytocic	1	0.1	0	0.0	1	0.0	0(0)	0.0
Other	0	0.0	0	0.0	0	0.0	0(0)	0.0
Total Inductions	74	10.5	83	4.8	157	6.5	3(1)	1.9
(Perinatal mortality over 1000 grams 5.8%)								
Total Elective Inductions	31	4.4	14	0.8	45	1.9	0(0)	0.0
B. Miscellaneous								
Decompression of hydrocephalus	1	0.1	0	0.0	1	0.0	0(0)	0.0
Forceps to after-coming head	10	1.4	4	0.2	14	0.6	0(0)	0.0
Manual removal of placenta, elective	64	9.1	99	5.8	163	6.7		
Manual removal of placenta, indicated	1	0.1	2	0.1	3	0.1		
Oxytocic stimulation of labor	1	0.1	8	0.5	9	0.4	0(0)	0.0
(Perinatal mortality over 1000 grams 1.0%.)								
Elective Oxytocic Stimulation	26	3.7	42	2.4	68	2.8	7(1)	10.3
Transfusion(s)	9	1.3	37	2.2	46	1.9		
Exploration of Uterus	4	0.6	9	0.5	13	0.5		
C. Episiotomies and lacerations								
Median	448	63.8	922	53.7	1370	56.7		
3rd degree lacerations	17	2.4	31	1.8	48	2.0		
4th degree lacerations	17	2.4	21	1.2	38	1.6		
Mediolateral	62	8.8	106	6.2	168	6.9		
3rd degree lacerations	2	0.3	2	0.1	4	0.2		
4th degree lacerations	0	0.0	0	0.0	0	0.0		
Total Episiotomies	546	77.8	1082	63.1	1628	67.3		
3rd degree laceration spontaneous, repair of	2	0.3	0	0.0	2	0.1		
4th degree laceration spontaneous, repair of	1	0.1	2	0.1	3	0.1		
Cervical laceration, repair of	3	0.4	5	0.3	8	0.3		
Vaginal laceration, repair of	10	1.4	10	0.6	20	0.8		

X. PUERPERAL MORBIDITY

	White		Non-White		Total	
	No.	%	No.	%	No.	%
One Day Fever	30	4.3	98	5.7	128	5.3
Standard Fever	32	4.6	150	8.7	182	7.5
Total	62	8.8	248	14.5	310	12.8
Infection						
Endometritis	12	1.7	59	3.4	71	2.9
Mastitis	0	0.0	0	0.0	0	0.0
Thrombophlebitis	0	0.0	3	0.2	3	0.1
Infected Wound	0	0.0	8	0.5	8	0.3
Peritonitis	0	0.0	0	0.0	0	0.0
Urinary tract	1	0.1	1	0.1	2	0.1
Respiratory disease	2	0.3	9	0.5	11	0.5
Other Complications						
Urinary Complication	1	0.1	2	0.1	3	0.1
Wound Dehiscence	0	0.0	1	0.1	1	0.0
Postspinal Symptoms	2	0.3	2	0.1	4	0.2
Total	3	0.4	5	0.3	8	0.3

XI. COMPLICATIONS

	White		Non-White		Total		Perinatal Mortality	
	No.	%	No.	%	No.	%	No.	%
A. Antepartum hemorrhage								
Placenta previa	1	0.1	8	0.5	9	0.4	1(0)	11.1
Abruptio placentae	4	0.6	19	1.1	23	1.0	8(2)	34.8
Rupture of uterus	0	0.0	1	0.1	1	0.0	0(0)	0.0
Traumatic	1	0.1	1	0.1	2	0.1	0(0)	0.0
Previous section, severe	1	0.1	0	0.0	1	0.0	1(0)	0.0
Previous section, incidental	0	0.0	0	0.0	0	0.0	0(0)	0.0
Other causes	3	0.4	12	0.7	15	0.6	1(0)	6.7
Total	10	1.4	41	2.4	51	2.1	11(2)	21.6
B. Postpartum hemorrhage								
Early	22	3.1	55	3.2	77	3.2		
Late	0	0.0	0	0.0	0	0.0		
Hematomata	0	0.0	0	0.0	0	0.0		
Total	22	3.1	55	3.2	77	3.2		
C. Anemia								
Less than 5 gm.	0	0.0	0	0.0	0	0.0	0(0)	0.0
5.0– 5.9 gm.	1	0.1	1	0.1	2	0.1	0(0)	0.0
6.0– 6.9 gm.	1	0.1	4	0.2	5	0.2	0(0)	0.0
7.0– 7.9 gm.	2	0.3	9	0.5	11	0.5	2(0)	18.2
8.0– 8.9 gm.	6	0.9	38	2.2	44	1.8	4(2)	9.1
9.0– 9.9 gm.	27	3.8	132	7.7	159	6.6	7(2)	4.4
10.0–10.9 gm.	75	10.7	341	19.9	416	17.2	16(5)	3.8
11 gm. and over	529	75.4	1167	68.0	1696	70.1	45(14)	2.7
Unknown	61	8.7	24	1.4	85	3.5	4(2)	4.7
Total	702	100.0	1716	100.0	2418	100.0	78(25)	3.2

XI. COMPLICATIONS (Cont.)

	White		Non-White		Total		Perinatal Mortality	
	No.	%	No.	%	No.	%	No.	%
D. Toxemia								
Pre-eclampsia—mild	30	4.3	124	7.2	154	6.4	6(0)	3.9
Pre-eclampsia—severe	2	0.3	10	0.6	12	0.5	2(0)	16.7
Eclampsia—antepartum	1	0.1	7	0.4	8	0.3	0(0)	0.0
Eclampsia—intrapartum	0	0.0	5	0.3	5	0.2	0(0)	0.0
Eclampsia—postpartum	2	0.3	1	0.1	3	0.1	0(0)	0.0
Total acute	35	5.0	147	8.6	182	7.5	8(0)	4.4
Chronic hyper. with toxemia	4	0.6	11	0.6	15	0.6	1(0)	6.7
Chronic hyper. without tox	9	1.3	87	5.1	96	4.0	4(0)	4.2
Total chronic	13	1.9	98	5.7	111	4.6	5(0)	4.5
Unclassified	0	0.0	2	0.1	2	0.1	0(0)	0.0
Total Toxemia	48	6.8	247	14.4	295	12.2	13(0)	4.4
E. Medical complications								
Heart disease	2	0.1	1	0.0	3	0.1	2(1)	100.0
No failure	1	0.1	0	0.0	1	0.0	0(0)	0.0
Failure	1	0.1	1	0.1	2	0.1	2(1)	100.0
Tuberculosis, pulmonary	0	0.0	5	0.3	5	0.2	1(1)	20.0
Viral pulmonary disease	2	0.3	1	0.1	3	0.1	0(0)	0.0
Other pulmonary disease	4	0.6	4	0.2	8	0.3	1(0)	12.5
Uncommon anemias	0	0.0	0	0.0	0	0.0	0(0)	0.0
Oliguria/anuria	0	0.0	0	0.0	0	0.0	0(0)	0.0
Pyelonephritis	3	0.1	4	0.2	7	0.3	0(0)	0.0
RH Negatives	102	13.7	102	5.9	204	8.9	7(0)	100.0
Rubella	0	0.0	0	0.0	0	0.0	0(0)	0.0
Diabetes	6	0.9	17	1.0	23	1.0	3(0)	13.0
Abnormal glucose tol. test	2	0.3	1	0.1	3	0.1	0(0)	0.0
F. Cord pathology								
Prolapse—Vaginal deliveries	2	0.3	3	0.2	5	0.2	2(1)	40.0
Prolapse—Abdominal deliveries	0	0.0	4	0.2	4	0.2	1(0)	25.0
Other	1	0.1	1	0.1	2	0.1	0(0)	0.0
G. Intrapartum fever	5	0.7	18	1.0	23	1.0	6(3)	26.1
H. Uterine dysfunction	19	2.7	59	3.4	78	3.2	4(0)	5.1
I. Labor over 20 hours—method of delivery								
Cesarean section	0	0.0	4	0.2	4	0.2	1(0)	25.0
Spontaneous	9	1.3	20	1.2	29	1.2	3(1)	10.3
Low forceps, elective	3	0.4	16	0.9	19	0.8	0(0)	0.0
Low forceps, indicated	1	0.1	1	0.1	2	0.1	0(0)	0.0
Mid forceps, elective	0	0.0	1	0.1	1	0.0	0(0)	0.0
Mid forceps, indicated	0	0.0	0	0.0	0	0.0	0(0)	0.0
Breech	0	0.0	1	0.1	1	0.0	0(0)	0.0
Other	0	0.0	2	0.1	2	0.1	0(0)	0.0
Total	13	1.9	45	2.6	58	2.4	4(1)	6.9
J. Shoulder dystocia	1	0.1	5	0.3	6	0.2	0(0)	0.0
K. Contracted pelvis	10	1.4	68	4.0	78	3.2	1(0)	1.3

XII. ABDOMINAL OPERATIONS

	White		Non-White		Total		Perinatal Mortality	
	Prim.	Repeat	Prim.	Repeat	Prim.	Repeat	Prim.	Repeat
A. Cesarean sections								
Low cervical	14	9	56	26	70	35	1 4	5 7
Low cervical and sterilization	4	7	3	25	7	32	0 0	3 1
Classical	0	1	13	0	13	1	30 8	0.0
Classical and sterilization	0	1	2	2	2	3	0.0	0.0
Extraperitoneal	0	0	0	0	0	0	0.0	0.0
Cesarean hysterectomy	0	0	0	0	0	0	0.0	0.0
Total Sections	18	18	74	53	92	71	5.4	4.2
Indications								
1. Pelvic contractions and mechanical dystocia								
Contracted pelvis	4	0	32	0	36	0	0.0	0.0
Large fetus	2	0	2	0	4	0	0.0	0.0
Uterine inertia	2	0	3	0	5	0	0.0	0.0
Malpresentation	2	0	4	0	6	0	0.0	0.0
Breech	1	0	0	0	1	0	0.0	0.0
Face	0	0	0	0	0	0	0.0	0.0
Brow	1	0	0	0	1	0	0.0	0.0
Transverse	0	0	4	0	4	0	0.0	0.0
Compound or other	0	0	0	0	0	0	0.0	0.0
Tumor blocking birth canal	0	0	1	0	1	0	0.0	0.0
Total	12	0	46	0	58	0	0.0	0.0
2. Previous uterine surgery								
Previous cesarean section	0	17	0	50	0	67	0.0	3.0
Previous myomectomy	0	0	0	0	0	0	0.0	0.0
Previous hysterotomy	0	0	0	0	0	0	0.0	0.0
Total	0	17	0	50	0	67	0.0	3.0
3. Hemorrhage								
Abruptio placentae	0	0	2	0	2	0	50.0	0.0
Placenta previa	0	0	3	2	3	2	33.3	0.0
Other	1	0	0	0	1	0	0.0	0.0
Total	1	0	5	2	6	2	33.3	0.0
4. Toxemia								
Pre-eclampsia	0	0	0	0	0	0	0.0	0.0
Eclampsia	0	0	1	0	1	0	0.0	0.0
Chronic hypertension and toxemia	0	0	0	0	0	0	0 0	0.0
Chronic hypertension	0	0	0	0	0	0	0.0	0.0
Total	0	0	1	0	1	0	0.0	0.0
5. Intercurrent diseases								
Diabetes	2	1	3	1	5	2	0.0	0.0
6. Miscellaneous Total								
Elderly Primagravida	0	0	1	0	1	0	0.0	0.0
Fetal distress	2	0	13	0	15	0	0.0	0.0
Prolapsed cord	0	0	3	0	3	0	33.3	0.0
Premature R. Membranes	1	0	1	0	2	0	0.0	0.0
Prev. Plastic vag.	1	0	2	0	3	0	0.0	0.0
Rh Incomp.	0	0	1	0	1	0	0.0	0.0
Postmortem	0	0	1	0	1	0	100.0	0.0
Hydrocephalus	0	0	1	0	1	0	100.0	0.0
Cong. Anomaly	1	0	0	0	1	0	0 0	0.0
Total	5	0	23	0	28	0	0.0	0.0

XII. ABDOMINAL OPERATIONS (Cont.)

	White	Non-White	Total
B. Cesarean hysterectomy	0	0	0
Indications for hysterectomy			
C. Puerperal hysterectomy	1	2	3
Indications			
Ruptured uterus	1	0	1
Hemorrhage	0	1	1
Myomata uteri	0	1	1
D. Laparotomies			
Advanced ectopic pregnancy	0	0	0
Rupture of uterus	1	0	1

XIII. DELIVERIES (INFANTS) WITH PREVIOUS SECTION

	White		Non-White		Total		Perinatal Mortality	
	No.	%	No.	%	No.	%	No.	%
Repeat section	18	2.6	53	3.1	71	2.9	3(0)	4.2
Vaginal deliveries								
Spontaneous	1	0.1	8	0.5	9	0.4	0(0)	0.0
Low forceps, elective	4	0.6	5	0.3	9	0.4	0(0)	0.0
Low forceps, indicated	0	0.0	0	0.0	0	0.0	0(0)	0.0
Mid forceps, elective	1	0.1	0	0.0	1	0.0	0(0)	0.0
Breech, spontaneous	0	0.0	0	0.0	0	0.0	0(0)	0.0
Breech, extraction	0	0.0	0	0.0	0	0.0	0(0)	0.0
Breech, decomposition & extraction	0	0.0	0	0.0	0	0.0	0(0)	0.0
Other (specify)	1	0.1	0	0.0	1	0.0	1(0)	100.0
Total	25	3.6	66	3.8	91	3.8	4(0)	4.4

XIV. THERAPEUTIC ABORTIONS

(NONE)

XV. STERILIZATION

	White	Non-White	Total
	No.	No.	No.
Type of Operation			
Tubal, puerperium	19	36	55
Tubal, not pregnant	0	0	0
Accompanying cesarean section	12	33	45
Accompanying therapeutic abortion	0	0	0
Hysterectomy, with cesarean section	0	0	0
Hysterectomy, not pregnant	0	0	0
X-ray	0	0	0
Total	31	69	100
Indications for Sterilization			
Multiple cesarean sections	7	24	31
Multiparity	22	43	65
Psychiatric	1	0	1
RH Incompatibility	0	1	1
Chronic Hypertension	0	1	1
Advanced Age	1	0	1
Total	31	69	100

XVI. ADULT DEATHS

Total Births	2418
Maternal deaths	3
Rate per 1000 births	1.24
Total Registered Births	2243
Maternal deaths (registered patients)	3
Rate per 1000 registered births	1.34

XVII. MALFORMATIONS

	White	Non-White	Total	Perinatal Mortality	
				No.	%
CNS System	1	0	1	0(0)	0.0
Face	4	5	9	0(0)	0.0
Cardiovascular System	2	2	4	0(0)	0.0
Digestive System	1	2	3	1(0)	33.3
Genito-Urinary System	9	19	28	1(1)	3.6
Skeletal System	4	27	31	1(0)	3.2
Integumentary System	0	3	3	0(0)	0.0
Miscellaneous	1	18	19	2(1)	10.5
Total	21	73	94	5(2)	5.3

XVIII. CAUSE OF PERINATAL DEATH

	White	Non-White	Total
Analgesia/anesthesia	0	0	0
Anomaly	1	2	3
Anoxia—Maternal	1	5	6
Anoxia—Obst	1	8	9
Anoxia—Unknown	9	28	37
Infection—Infant	1	5	6
Infection—Maternal	0	1	1
Isoimmunization	0	1	1
Respiratory Disease	2	13	15
Trauma	0	0	0
Other	0	0	0
Total	15	63	78

XIX. INFANTS DELIVERED

A. Total Live Births According to Weight and Condition at Discharge

Birth Weight Grams	White			Non-White			Total		
	Live Births	Died	%	Live Births	Died	%	Live Births	Died	%
500–999	5	2	40.0	17	11	64.7	22	13	59.1
1000–1499	1	1	100.0	31	15	48.4	32	16	50.0
1500–1999	6	1	16.7	57	2	3.5	63	3	4.8
2000–2499	55	0	0.0	179	4	2.2	234	4	1.7
2500 and over	625	1	0.2	1404	3	0.2	2029	4	0.2
Total	692	5	0.7	1688	35	2.1	2380	40	1.7

B. Total Stillbirths According to Weight

Birth Weight Grams	White			Non-White			Total		
	Total Births	Still-births	%	Total Births	Still-births	%	Total Births	Still-births	%
500–999	6	1	16.7	28	11	39.3	34	12	35.3
1000–1499	2	1	50.0	36	5	13.9	38	6	15.8
1500–1999	9	3	33.3	59	2	3.4	68	5	7.4
2000–2499	58	3	5.2	182	3	1.6	240	6	2.5
2500 and over	627	2	0.3	1411	7	0.5	2038	9	0.4
Total	702	10	1.4	1716	28	1.6	2418	38	1.6

XIX. INFANTS DELIVERED (Cont.)

C. Total Perinatal Deaths According to Weight

Birth Weight Grams	White			Non-White			Total		
	Total Births	Perinatal Deaths	%	Total Births	Perinatal Deaths	%	Total Births	Perinatal Deaths	%
500–999	6	3	50.0	28	22	78.6	34	25	73.5
1000–1499	2	2	100.0	36	20	55.6	38	22	57.9
1500–1999	9	4	44.4	59	4	6.8	68	9	11.8
2000–2499	58	3	5.2	182	7	3.8	240	10	4.2
2500 and over	627	3	0.5	1411	10	0.7	2038	13	0.6
Total	702	15	2.1	1716	63	3.7	2418	78	3.2

OBSTETRICAL DEATHS

E. B., UH, #32-46-13, a 19 yr. old C.F. para 0-0-0-0, admitted 7 27 66 with premature rupture of membranes. She delivered a full term female elective forceps, had a prolonged second stage. The patient expired within 24 hours with cardiac arrest. No autopsy was obtained.

E. F., UH, #22-91-10, a 37 yr. old C.F. para 7-0-1-1, admitted 7 18 66 with premature rupture of membranes and fever. Delivered a stillborn female by cesarean section. The patient expired within 24 hours with embolism cardiovascular.

B. P., UH, #30-62-77, a 28 yr. old C.F. para 1-2-1-0, admitted 10 4 66 with premature rupture membranes. She spontaneously delivered a premature male. Patient then went into shock during labor and had an evacuation of hematoma of the uterine segment. She expired the same day of septic shock with cardiac arrest.

GYNECOLOGIC REPORT

I. DISCHARGES PER PATIENT

	1	2	3	4	5	Total
Number of patients	886	107	13	7	1	1014

II. GENERAL DISCHARGE TYPE

	Ward	Private	Total
Number of discharges	769	403	1172
A. Gynecologic benign	445	265	710
1. Surgical	352	217	569
a. Minor, single	128	113	241
b. Minor, multiple	2	0	2
c. Major, single	204	94	298
d. Major, multiple	17	10	27
2. Non-operative	72	21	93
3. For diagnosis only	22	27	49
B. Gynecologic cancer	137	62	199
C. Pregnancy complications	183	67	250
D. Miscellaneous	4	9	13

III. DEATHS

	Ward	Private	Total
A. Operative	0	0	0
B. Non-operative	0	0	0
C. Diagnosis only	0	0	0
D. Cancer	7	1	8
E. Pregnancy complications	0	0	0
F. Miscellaneous	0	0	1
Total	7	1	8

IV. TRANSFERS

	Ward	Private	Total
Number	11	2	13

V. PRIMARY AND SECONDARY GYNECOLOGIC DIAGNOSIS

A. Vulva

Diagnosis	Primary	Secondary
Abscess, Bartholin's gland	3	0
Cyst, Bartholin's gland	4	2
Hymen, Imperforate	1	0
Leukoplakia	1	0
Vulvitis, Acute	2	1
Vulvitis, Chronic	0	1
Other	7	0
Condylomata	1	1
Senile Atrophy	0	1
Anomaly	1	1
Endometriosis	1	0
Total	21	7

B. Vagina

Diagnosis	Primary	Secondary
Anomaly	2	1
Cyst, inclusion	1	0
Cystocele	29	66
Fistula, rectovaginal (p.o.)	1	0
Fistula, rectovaginal trauma	3	0
Rectocele	6	53
Tear, 4th degree	2	0
Vaginitis, fungus	1	2
Other	0	1
Urethrocele	0	3
Incomplete tear	4	0
Total	49	126

C. Cervix

Diagnosis	Primary	Secondary
Cervicitis, acute	2	22
Cervicitis, chronic	19	251
Cyst, Nabothian	0	11
Polyp	7	7
Prolapse, stump	1	0
Basal cell hyperplasia	0	2
Stenosis	2	1
Scar following operation	0	1
Myoma	0	3
Total	31	298

D. Uterus

Diagnosis	Primary	Secondary
Adenomyosis	4	8
Anomaly	1	3
Carcinoma, metastatic	0	1
Endometritis, acute	6	5
Endometritis, chronic	0	3
Endometrium, atrophic	2	23
Endometrium, hyperplastic	9	19
Endometrium, proliferative	0	102
Endometrium, secretory	0	50
Fibromyomata	136	52
Foreign body	0	1
Metritis, acute	1	1
Metritis, chronic	0	1
Parametritis	0	5
Perforation, instrumental	0	1
Polyp, endometrial	14	20
Pregnancy, intrauterine	0	9
Prolapse	47	13
Retroversion	1	0
Retained secundines	0	3
Hyperplasia	0	1
Subinvolution of placental site	0	2
Placental tissue, abortion	0	1
Fibrosis	1	2
Torsions of subserous myoma	0	1
Inversion	0	1
P.P. Hemorrhage	0	1
Total	222	328

E. Tubes

Diagnosis	Primary	Secondary
Abscess, tubo-ovarian (intact)	19	4
Abscess, tubo-ovarian (rupture)	5	1
Endometriosis	0	3
Hematosalpinx	0	1
Hydrosalpinx	5	13
Pyosalpinx	2	10
Salpingitis, acute	17	15
Salpingitis, chronic	9	61
Perisalpingitis, acute	1	0
Perisalpingitis, chronic	0	3
Primary carcinoma, tubes	0	1
Total	58	112

V. PRIMARY AND SECONDARY GYNECOLOGIC DIAGNOSIS (Cont.)

F. Ovary

Diagnosis	Primary	Secondary
Cyst, corpus luteum	11	24
Cyst, dermoid	8	4
Cyst, follicular	3	20
Cyst, other	0	1
Cyst, paroophron	2	3
Cyst, simple	3	2
Cyst, undetermined	9	5
Cystadenoma, pseudomucinous	1	0
Cystadenoma, serous	6	6
Endometriosis	2	1
Fibroma	1	0
Oophoritis, acute	0	5
Oophoritis, chronic	1	8
Other	0	3
Ovaries, poly cystic (S.L. Disease)	10	0
Perioophoritis	0	1
Adenofibroma of ovary	1	1
Abscess	0	1
Atrophy	0	2
Unlisted tumors of ovary	0	1
Hemorrhage	0	1
Total	58	89

G. Other Gynecologic Diagnosis

Diagnosis	Primary	Secondary
Abscess, pelvis	5	0
Amenorrhea, hypothalamic	3	0
Bleeding, functional uterine	115	7
Bleeding, postmenopausal	43	1
Endometriosis, pelvis	1	2
Infertility	1	5
Mass, adnexal	16	3
Pain, pelvic	2	1
Peritonitis, pelvic	0	1
Smear, Pap. inconclusive	1	2
Sterility	3	0
Sterilization	64	7
Smear, Pap. positive	1	2
Post biopsy bleeding	1	0
Total	256	31

VI. CANCER (Based on Patients)

A. Vulva

1. Diagnosis	Number of Patients	Irradiated	Operations	Irradiated and Operated	Medical
Adenocarcinoma	0	0	0	0	0
Epidermoid	3	0	3	0	0

2. Complications	Number
Carcinoma metastatic to lymph nodes out of pelvis	1
Carcinoma metastatic to lymph nodes of pelvis	1

B. Vagina

1. Diagnosis	Number of Patients	Irradiated	Operations	Irradiated and Operated	Medical
Epidermoid	2	0	0	1	1
Adenocarcinoma	0	0	0	0	0

VI. CANCER (Based on Patients) (Cont.)

C. Cervix

1. Diagnosis	Number of Patients	Irradiated	Operations	Irradiated and Operated	Medical
Adenocarcinoma	4	1	2	0	1
Carcinoma in situ	29	1	28	0	0
Squamous cell	55	28	17	0	10
Total	88	30	47	0	11

2. Complications	Number
Cystitis, chronic	3
Fistula, rectovaginal, radium	5
Fistula, vesicovaginal tumor	4
Hydronephrosis	4
Hydroureter	4
Lymph nodes in groin, metastatic	7
Lymph nodes in pelvis, metastatic	7
Lymph nodes out of pelvis, metastatic	7
Pregnancy	2
Proctitis, chronic	1
Stricture, rectal	1

D. Uterus

1. Diagnosis	Number of Patients	Irradiated	Operations	Irradiated and Operated	Medical
Adenocanthoma	0	0	0	0	0
Adenocarcinoma	14	0	7	2	5
Mixed Mesodermal	1	0	0	1	0
Sarcoma	7	0	2	0	5
Undifferenitiated	0	0	0	0	0
Total	22	0	9	3	10

2. Complications	Number
Lymph nodes, groin metastatic	0
Lymph nodes in pelvis, metastatic	1
Lymph nodes out of pelvis, metastatic	3

E. Tubes

1. Diagnosis	Number of Patients	Irradiated	Operations	Irradiated and Operated	Medical
Carcinoma	0	0	0	0	0

F. Ovary

1. Diagnosis	Number of Patients	Irradiated	Operations	Irradiated and Operated	Medical
Adenocarcinoma	3	2	1	0	0
Brenner	0	0	0	0	0
Cystadenocarcinoma pseudomucinous	6	2	0	0	4
Cystadenocarcinoma, serous	4	0	0	0	4
Granular cell	1	0	1	0	0
Teratoma	2	0	1	0	1
Undifferentiated	1	0	0	0	1
Total	17	4	3	0	10

VI. CANCER (Based on Patients) (Cont.)

F. Ovary (Cont.)

2. Complications	Number
Lymph nodes in pelvis, metastatic	5
Lymph nodes out of pelvis, metastatic	6

G. Unknown Source

Diagnosis	Number of Patients
	0

VII. PREGNANCY COMPLICATIONS

Diagnosis	Number of Patients
Abortion, incomplete	187
Cervix, incompetent	0
Hemorrhage, postpartum, late	3
Infection, puerperal	1
Mole, hydatid	2
Pregnancy, intrauterine	28
Pregnancy, tubal ruptured, internal	3
Pregnancy, tubal unruptured	1
Pregnancy, tubal ruptured, external	17
Retained Secundines	1
Subinvolution of placental site	5
Ovarian pregnancy	1
Abdominal pregnancy	1
Total	250

VIII. OTHER SYSTEMS—DIAGNOSIS

A. Rectum

Diagnosis	Primary	Secondary
Stricture	0	2
Others	0	3
Total	0	5

B. Urethra

Diagnosis	Primary	Secondary
Diverticulum	1	2
Urethritis, acute	0	1
Urethritis, chronic	1	5
Incontinence	0	1
Stricture	1	2
Anomaly	0	1
Total	3	12

C. Bladder

Diagnosis	Primary	Secondary
Carcinoma, metastatic	0	1
Carcinoma, primary	2	0
Cystitis, acute	1	0
Cystitis, chronic	2	8
Other	3	5
Anomaly	0	7
Total	8	21

D. Ureters

Diagnosis	Primary	Secondary
Anomaly	0	1
Hydroureter	0	3
Total	0	4

VIII. OTHER SYSTEMS—DIAGNOSIS (Cont.)

E. Kidneys

Diagnosis	Primary	Secondary
Anomaly	2	2
Hydronephrosis	0	3
Other	0	1
Total	2	6

F. Abdominal Diseases

Diagnosis	Primary	Secondary
Adhesions, peritoneal	1	20
Appendicitis	4	0
Ascites	1	0
Endometriosis, site unspecified	1	0
Hematoma, ant. abd. wall, inf.	1	1
Hernia, incisional	0	1
Ileus, paralytic	0	1
Lymph nodes out of pelvis	0	2
Miscellaneous	2	0
No disease	3	0
Obstruction, small intestine	2	2
Peritonitis, abdominal	0	6
Total	15	33

IX. OPERATIVE PROCEDURES

A. Vulva

Operations	Number of Patients
Barhtolin Gland, excision	7
Bartholin Gland, I&D	3
Biopsy	2
Other	8
Vulvectomy, complete	1
Vulvectomy, partial	0
Vulvectomy, radical	2
Hymenotomy	1
Total	24

B. Vagina

Operations	Number of Patients
Biopsy	7
Colpectomy, partial	2
Colpeperineorrhaphy	4
Colpoplasty, anterior	21
Colpoplasty, posterior	6
Colpoplasty, anterior and posterior	47
Colpoporrhaphy	8
Colpotomy, diagnostic	31
Colpotomy, drainage	1
Other	19
Radioactive substances, insertion of	110
Perineorrhaphy	1
Total	257

C. Uterus and Cervix

Operations	Number of Patients
Cervix, biopsy	258
Conization	40
Dilation, cervix	1
D&C, uterus, diagnostic	337
D&C, uterus, incomplete abortion	162
Excision, local, cervix	0
Hysterectomy, radical and lymph node	4
Hysterectomy, subtotal	7
Hysterectomy, total abdominal	184
Hysterectomy, total vaginal	54
Hysteromyomectomy	9
Hysteropexy, Manchester type	1
Hysteropexy, other types	2
Other	3
Radioactive sub. inserted into cervix	16
Radioactive sub. inserted into uterus	65
Trachelectomy	2
Trachelorrhaphy	1
Uterus, insuffation of	5
Hysterotomy	1
Hysterogram	2
Total	1154

IX. OPERATIVE PROCEDURES (Cont.)

D. Tubes

Operations	Number of Patients
Salpingectomy, unilateral, partial	9
Salpingectomy, bilateral, partial	70
Salpingectomy, unilateral, complete	7
Salpingectomy, bilateral, complete	5
Salpingo-oophorectomy, unilateral	26
Salpingo-oophorectomy, bilateral	12
Salpingoplasty	3
Other operation on fallop. tubes	1
Total	133

E. Ovary

Operations	Number of Patients
Excision, local lesion	5
Oophorectomy, unilateral, complete	4
Other	19
Drain of ovary, cyst, abscess, etc	1
Oophorectomy, unilateral, partial	10
Oophorectomy, bilateral, complete	4
Total	43

F. Urinary System

Operations	Number of Patients
Bladder, biopsy	6
Cystectomy	4
Cystorrhaphy	4
Cystoscopy, diagnostic	76
Cystoscopy, therapeutic	9
Ileal loop	2
Nephrectomy	1
Ureterostomy	2
Total	104

G. Abdominal and Others

Operations	Number of Patients
Abdominal wall, second suture of	2
Adhesions, lysis of	17
Appendectomy	56
Colostomy	3
Dissection, radical groin	4
Herniorrhaphy	5
Laparotomy, exploratory	79
Lymph node, biopsy of	8
Mesentery, biopsy of	2
Miscellaneous	39
Peritoneocentesis	14
Proctoscopy	21
Sympathectomy	2
Ligation of vein	3
Omentectomy	1
Culdoscopy	4
Ileostomy	1
Total	261

H. Irradiation

Type	No. of Times
Isotopes	1
Other	8
Radium	37
Total	46

X. MORBIDITY AND COMPLICATIONS OF OPERATIONS

A. Minor Single

1. Total	Number	Morbidity	% Morbidity
	549	13	2.4

2. Causes of Morbidity	Number
Cause unknown	13

X. MORBIDITY AND COMPLICATIONS OF OPERATIONS (Cont.)

B. Minor Multiple

1. Total	Number	Morbidity	% Morbidity
	11	1	9.1

2. Causes of Morbidity	Number
Cause unknown	1

3. Complications	Number
Endometritis	1
Total	1

C. Major Single

1. Total	Number	Morbidity	% Morbidity
	405	87	21.5

2. Causes of Morbidity	Number
Abdominal wound infection	11
Cause unknown	19
Pelvic abscess	1
Peritonitis	6
Pulmonary	5
Thrombophlebitis	1
Urinary tract	33
Other	9
Abdominal abscess	2
Total	87

3. Complications	Number
Paralytic ileus	9
Respiratory disease	5
Secondary anemia	4
Thrombosis	1
Urinary retention	3
Wound infection	4
Other	2
Total	28

D. Major Multiple

1. Total	Number	Morbidity	% Morbidity
	39	10	25.6

2. Causes of Morbidity	Number
Abdominal wound infection	4
Causes unknown	5
Urinary tract	1

3. Complications	Number
Paralytic ileus	1
Postoperative hemorrhage	3
Secondary anemia	2
Wound breakdown	1
Total	7

DEATHS

S. H. UH-30-66-14, a 31 yr. old W.F. ad. 1/18/66 Epidermoid Carcinoma. Died 1/26/66 of Carcinoma of the Cervix, stage III with extension of the vagina.

M. B. UH-32-68-40, a 18 yr. old C.F. ad. 4/20/66 Malignant Teratoma of the left Ovary. Died 5/13/66 of Malignant Sarcoma of the left Ovary with distant bleeding.

M. S. UH-32-66-95, a 67 yr. old W.F. ad. 6/8/66 Mixed Mesodermal Tumor of the Endometrium. Died 6/17/66 of Anoxia.

D. H. UH-31-62-10, a 56 yr. old C.F. ad. 9/9/66 Carcinoma of the Cervix. Died 9/10/66 of Carcinoma of the Cervix.

P. W. UH-31-76-92, a 43 yr. old C.F. ad. 10/6/66 Epidermoid Carcinoma. Died 10/21/66 of Uremia.

E. K. UH-30-45-27, a 47 yr. old W.F. ad. 11/10/66 Terminal Carcinoma of the Cervix. Died 11/22/66 of complications secondary to Terminal Carcinoma of the Cervix, stage IV.

R. H. UH-34-02-42, a 55 yr. old C.F. ad. 12/27/66 Carcinoma of the Vulva. Died 12/30/66 of Cardiac Arrest.

MEDICAL SCHOOL SECTION

Dean's LETTER

Dear Members of the Medical Alumni, Students and Friends of the Medical School:

I am unable to bring you news of the legislative action on the Medical School's request for capital improvement funds for the addition to the Basic Science building. No action has been taken at the time this letter goes to press.

The Admissions Committee has been diligent in its selection of students for admission and it appears that outstanding students have been selected for beginning their studies at Maryland in September 1968.

The curriculum will be further revised in June of this year so that the changes made in the first two years will be carried forth into the third and fourth years starting in September 1968. These changes should give the Medical School a much better co-ordinated and integrated teaching program. There is much more leeway for independent career development on the part of the students with some opportunity for backgrounds in family practice as well as specialty and research interests.

Sincerely,

William S. Stone, M.D.
Dean

University Hospital Annual Report

The BULLETIN is pleased to publish a portion of the annual report of the University of Maryland, in which hospital director Dr. George H. Yeager has outlined its achievements for the year ending June 30, 1967.

Copies of the full report, giving detailed statistics, are available through the office of the director.

Report of the Director

This year final plans were completed for construction of the new Center of Trauma. The bid has been awarded and construction is to start the 1st of July, 1967. It is anticipated that this four-story addition to the Hospital will be completed in approximately fifteen to eighteen months. This Center of Trauma will provide a new and unique facility for the study of trauma and shock, and will certainly be a landmark institution in this aspect of Medicine. The North Hospital Building is entering its final stages of planning and construction is scheduled to commence in mid '68.

The Hospital continued to grow both in personnel and in intensified professional care of patients. The former Division of Oral Surgery was made a Department. This, together with the addition of Neurology as a Department, has increased the number of Clinical Departments within the Hospital to twelve.

It is a tribute to the staff that despite the frustrations caused by overcrowded conditions and personnel shortages, the Hospital continued to maintain a high level of performance.

Numberous Federal grants were received and other Federal grants were renewed for continued research. With the expansion of facilities it is anticipated that additional research projects will be undertaken. The Hospital made substantial expenditures in capital equipment and several major Hospital renovations were completed. The Clinical Laboratories were expanded and renovated and various automated equipment has been purchased in order to speed up and provide more laboratory testing.

The main entrance of the first floor of the Hospital has been renovated with the expansion of the patient admission activity and the addition of a Chapel. Plans are underway for a major expansion of the Kitchen and Cafeteria. Money has been requested by the Hospital to renovate and double the present capacity of these facilities.

The advent of Medicare and Medicaid has increased the burden on the already overcrowded facilities of the institution. This is particularly true of the ambulant clinics. The shortage of nurses continues to plague the Hospital as it does most hospitals in the nation. Every effort is being made to recruit additional nurses. Salary increases, improved working conditions, and fringe benefits are proving to be effective. It is anticipated in the coming year that all Hospital activities will be able to function at their maximum.

There has been an increased utilization of diagnostic facilities as shown by the number of X-ray examinations, EKG, EEG, cardio-pulmonary and cystoscopic tests performed.

GEORGE H. YEAGER, M.D.
Director

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ALUMNI ASSOCIATION SECTION

President's Letter

Dear Fellow Alumni:

As you know, the only excuse for the existence of any alumni association is its contributions to the Alma Mater. We have been made aware of the need for individual financial aid to institutions of higher learning, but there are some very important contributions alumni can make that are rarely brought to mind.

With the ever increasing federalization of our profession, it is mandatory that each of us exert his maximum political influence to assure the freedom for all to practice medicine at its best.

Also, through our intimate knowledge of intelligent and studious young people, earnestly desiring to become physicians, we can and should sponsor their applications and thus aid the Admissions Committee to obtain superior medical students.

Finally, the everyday performance of our responsibilities as physician, teacher, scientist, or civic leader will always serve as the intimate test of this medical school and thus, our independent actions should be the most significant contributions of the University of Maryland Medical Alumni Association.

June 5, 1968

Sincerely yours,

John O. Sharrett, M.D.

John O. Sharrett, M.D.
President, Medical Alumni Association

Dr. Kardash Reports on Meetings of Board of Directors, Medical Alumni Association

September 6, 1967

This being the first fall meeting of the Medical Alumni Association's Board of Directors, Dr. John O. Sharrett, President, reported informally on work done during the summer. In accordance with the new membership ruling included in the Alumni Association's revised constitution—ratified at the annual business meeting on Alumni Day, June 1, 1967—invitations were sent to 640 hospital faculty and staff members who are not graduates of the University of Maryland.

Plans were described for establishing *Curriculum Vitae* as a vital part of the biographical records kept for each alumnus (later to be recorded on IBM cards), and a form chosen for this purpose will be circulated.

Needs of the BULLETIN and how the Alumni Association can help meet these needs were discussed. Subscription price for the BULLETIN will remain at $3.00 per member; however, in May, 1968, an additional $1.00 will be contributed for each paid Alumni Association membership. Cost of subscriptions sent to members of the 2 most recent graduating classes and to all alumni graduated more than 50 years ago will be borne by the Alumni Association.

October 4, 1967

At the October meeting plans were made for the University of Maryland reunion at the District of Columbia Scientific Assembly and Dr. William Holbrook of College Park was placed in charge. A progress report was given concerning the meeting to be held October 21-23, 1967, at Brunswick Hospital, Amityville, New York. The program was reviewed and approved by members of the Board.

Dr. Sharrett announced that the Medical Alumni Association has been promised Davidge Hall as its home base. Costs of rehabilitating the building were discussed.

Plans were reviewed for Alumni Day, June 6, 1968, and for the third Maryland Medical Reunion, which will also be held then, with hospitality night planned for June 5. Respective departments are formulating their scientific programs. Final discussion concerned the new statement envelope, to be decided upon shortly.

November 1, 1967

Reports were presented on the meeting at Brunswick Hospital in New York; and finalized plans were announced for the Medical Society of the District of Columbia meeting. Drs. Elkins and Stone speak at the luncheon; Dr. Sharrett will also attend. Reports on the development of plans for Alumni Day and the Maryland Medical Reunion indicated that satisfactory progress is being made.

The meeting concluded with discussion of the restoration of Davidge Hall, the consensus being that the hall should be made a State Historical Monument and that at least one lecture for each class should be held in the building annually. Dr. Sharrett will speak with President Elkins regarding the project and will report at the next meeting whether the Alumni Association has authorization to proceed with restoration plans, and possible means for collecting monies required for such restoration.

December 6, 1967

Suggestions were made regarding the content of a form to be sent each alumnus every fifth year to obtain current information for the new *Curriculum Vitae.*

Dr. Mays reported on the success of the New York area reunion held at Amityville, New York. The meeting prompted several contributions, and Dr. Mays recommended that similar area reunions to be held in the future.

The new statement envelope chosen by Dr. Kardash's committee was approved. Dr. Sharrett reported on the progress of plans for Alumni Day, 1968.

The reunion held during the meeting of

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Class NOTES

Elsewhere in this edition you will find a "tear out" page, for reporting Alumni News to the BULLETIN. *This is not an idle gesture.*

Your achievements, fellow alumnus, are of interest to your classmates. They constitute a reward to the faculty, are a challenge to the younger physicians, and are an item of prestige for the University. Please cooperate with us by forwarding news of yourself or any alumnus to the BULLETIN. *Thank you.*

A host of replies in response to the Medical Alumni Association's questionnaire have been received, many of them being of great interest. Some will be subsequently published in the BULLETIN of the School of Medicine so as to acquaint the many alumni of this old and traditional institute of the accomplishments of its alumni who are practicing in many parts of the world. Typical of one of the responses is a biography submitted by one of the School's oldest and most active graduates, Dr. John Robert Lowery of the Class of 1904.

CLASS OF 1904

Dr. Lowery's Interesting Career

A native of Davie County, North Carolina, Dr. Lowery completed his school at the Harmony Cool Springs and at Mecksville. After attending the University of North Carolina, he completed his medical studies at the School of Medicine in 1904 and returned to Cooleemee, North Carolina, where he entered into the practice of general medicine at Cool Springs.

In 1910 he decided to specialize in gastroenterology and so studied with Dr. Julius Friedenwald for one year. Following this sojourn, he went to Germany and studied there under Dr. Ewald at the University of Berlin. After a period of two years he returned to the United States and then located in Raleigh, North Carolina, where he specialized in diseases of the stomach for more than seventeen years. Following this, he returned to Salisbury, North Carolina, and opened a private general hospital which he conducted for almost two decades.

Dr. Lowery is proud to claim himself as a Tarheel. He is a member of the Methodist Church and of the Salisbury Kiwanis Club. He is an honorary member of the Rowan-Davie Medical Society, the State Medical Society and the American Medical Association. He has owned 16 farms including large peach and apple orchards in the Sandhills and supervised the operation of these. Now, at the age of 86 and retired, he has overcome a number of physical ailments and has shown steady improvement. Now he has become active in the raising of quail, turkeys, chickens and other poultry at his home at Milford Mills. He also has a hobby of making grandfather clocks. A frequent speaker at civic clubs and medical societies, Dr. Lowery remains active and continues his life as a dynamic member of his community and of the medical environment. He is recently the author of *Memoirs of a County Doctor,* however this opus has not as yet appeared in press.

CLASS OF 1937

Dr. Eugene S. Bereston, associate professor of dermatology in the School of Medicine, was recently appointed Chief of Dermatology at Mercy Hospital, Baltimore.

CLASS OF 1943

Dr. F. Mason Sones, Jr., of the Cleveland Clinic Foundation addressed the annual meeting of the Medical and Chirurgical Faculty of Maryland at the Alcazar in Baltimore on the afternoon of Wednesday, April 17, 1968. Dr. Sones's topic was "Roentgen-cine Coronary Arteriography in Evaluating Medical and Surgical Therapy for Coronary Atherosclerosis."

CLASS OF 1947

Dr. B. Stanley Cohen has been named chief of staff for the new $1,750,000 Ansel

and Ellen Schoeneman Rehabilitation Center at Sinai Hospital in Baltimore. The Center, which was opened on December 27, 1967, is the most modern and comprehensive facility of its kind in the state.

Dr. Cohen's interest in arthritis prompted him to institute an arthritis clinicc, which he conducted for ten years at Sinai. The hope of expanding these efforts into a department of physical medicine and rehabilitation led him to give up private practice in 1963 and devote the next two and one-half years to studying rehabilitative medicine in Dallas at Baylor University Medical Center and at Parkland Memorial Hospital.

In 1965 Dr. Cohen returned to Sinai to establish the Department of Physical Medicine and prepare the program for the new Schoeneman Center. The Center is equipped to treat 300 outpatients daily, and is staffed by specialists in medical evaluation, physical and occupational therapy, speech and hearing services, psychology, rehabilitation nursing, vocational counseling and social service.

CLASS OF 1952

Dr. John L. Watters of Huntington, New York, has been named corporate medical director of the Becton, Dickinson Company of Rutherford, New Jersey. In his new position Dr. Watters will hold medical responsibility for all the company's product lines.

A graduate of the University of North Carolina and of the School of Medicine, Dr. Watters has published numerous research papers on bacteriology and its related fields. He has also served as medical director at the Charles Pfizer Laboratories Division and at the J. B. Roerig Division of that company. Dr. Watters is a former president of the Association of Medical Directors, and a member of the American Medical Association, the American Association for the Advancement of Science and the New York Medical Society.

CLASS OF 1961

Dr. Thomas McGeoy, Jr., of Oakland, California, writes: "To plagiarize Mark Twain, 'reports of my death have been greatly exaggerated.' "

The BULLETIN is more than happy to acknowledge its error.

Continued from page xxx

the Medical Society of the District of Columbia was reported to have been a great success. Thanks to the able chairmanship of Dr. William Holbrook, Jr., of the class of 1945, attendance surpassed that at the regular meetings of the local alumni group. Through the Alumni Office's new IBM listing, it is now hoped that the local alumni club president will be able to reach many more area alumni.

THEODORE KARDASH, M.D.
Secretary

Deaths

CLASS OF 1897 P&S

Dr. Claude D. J. MacDonald, of MacDonald Drive, Norfolk, Virginia, died June 3, 1967. Dr. MacDonald was 96.

CLASS OF 1901 BMC

Dr. Robert Walker Love of Moorefield, West Virginia, died in January, 1968. Dr. Love was 94.

CLASS OF 1901 P&S

Dr. Charles W. Daly of 139 Warrenton Avenue, Hartford, Connecticut, died August 31, 1967, at the age of 84.

Dr. George F. Grisinger of 2861 Piedmont Road, Charleston, West Virginia, died December 20, 1967, at the age of 81.

CLASS OF 1902

Dr. Albert Singewald, 93 and the oldest practicing physician in Baltimore City, died on March 2, 1968, after a brief illness.

Dr. Singewald was active until a few weeks before his death and continued to see patients at his office on North Avenue. At the time of his death, he was caring for at least fifteen Baltimore families, six of whom he had been treating for three generations.

A native of Baltimore and a graduate of Cornell University, Dr. Singewald began practicing shortly after his graduation from the School of Medicine. In World War I, he was Commander of the 313th Army Field Hospital in France, reaching the rank of colonel.

CLASS OF 1903 BMC

Dr. John Evans, Sr., a pioneer radiologist in Baltimore and former chief of radiology at the Maryland General Hospital, died at the Long Green Nursing Home on June 28, 1967. Dr. Evans was 88.

A native of Lock Haven, Pennsylvania, Dr. Evans graduated from the Baltimore Medical College which then merged with the University of Maryland in 1913. After interning at Maryland General Hospital, he spent two years in general practice in southwestern Pennsylvania, when he returned to Baltimore and to private practice. During World War I, he served in France with Base Hospital 42, a University of Maryland unit. For 35 years and until he resigned in 1948, Dr. Evans served as radiologist in chief at the Maryland General Hospital. He retired in 1958.

CLASS OF 1904 P&S

Dr. Morris D. Cohen of 1534 E. Speedway, Tucson, Arizona, died October 24, 1967. Dr. Cohen was 86.

CLASS OF 1906

Dr. Romulus L. Carlton of Reynolds Park Road, Winston-Salem, North Carolina, died September 8, 1967, at the age of 86.

CLASS OF 1906 BMC

Dr. Linn F. Playse of 4340 Juanita Way, South, St. Petersburg, Florida, died on January 16, 1968.

Dr. Jacob Roemer of 365 S. Westgate Avenue, Los Angeles, California, died June 22, 1967. Dr. Roemer was 84.

CLASS OF 1907 BMC

Dr. Peter C. Mikkelsen of 3671 Country Club Drive, Redwood City, California, died in January, 1965.

CLASS OF 1907 P&S

Dr. Walter A. Carr of War, West Virginia, died October 6, 1967.

CLASS OF 1908 BMC

Dr. Otto George Matheke, Sr. of 108 E. Munn Avenue, East Orange, New Jersey, died on June 16, 1967. Dr. Matheke was 84.

Dr. Thomas W. Stevenson of 4421 Montaire Avenue, Long Beach, California, died March 16, 1967. Dr. Stevenson was 88.

CLASS OF 1908 P&S

Dr. Ivan Emerson Pratt of Millerton, Pennsylvania, died January 30, 1968. Dr. Pratt was 83.

CLASS OF 1909

Dr. William G. C. Hill of 1309 3rd Street, Moundsville, West Virginia, died recently.

CLASS OF 1909 P&S

Dr. William Garfield Phillips of Skiatook, Oklahoma, died July 12, 1967. Dr. Phillips was 89.

CLASS OF 1910

Dr. Herber M. Foster died at his home, 2220 Westchester Avenue, Catonsville, Maryland, on Thursday, November 23, 1967. Dr. Foster was 79.

A native of Baltimore, Dr. Foster interned at the Baltimore City Hospitals entering private practice in 1913. He served actively on the staffs of the Maryland General and University Hospitals and for a number of years maintained offices at 2824 St. Paul Street and at a farm he owned in Howard County. During World War I, he served overseas in the Army Medical Corps. In 1940, he was appointed by Mayor Howard W. Jackson to serve as one of seven doctors comprising a newly formed disability compensation board designed to centralize the handling of city employees who were injured during the performance of official duties.

Dr. Louis Rubin of 10510 Euclid Avenue, Cleveland, Ohio, died July 2, 1967, at the age of 79.

CLASS OF 1911

Dr. Java C. Wilkins of Haw River, North Carolina, died on July 7, 1967. Dr. Wilkins was 81.

CLASS OF 1912 BMC

Dr. Charles Jacob Greenstein of 300 Main Street, New Britain, Connecticut, died June 30, 1967. Dr. Greenstein was 80.

Dr. Simon Geilech Lenzner of 187 Waterman Street, Providence, R. I., died recently.

CLASS OF 1913

Dr. William T. Martin of 605 McDaniels Avenue, Greenville, South Carolina, died April 7, 1967. Dr. Martin was 77.

CLASS OF 1913 P&S

Dr. Charles William Finnerty of 440 Broadway, Somerville, Massachusetts, died on September 9, 1967, at the age of 78.

CLASS OF 1915

Dr. William H. Jenkins of 2024 R Street, N.W., Washington, D. C., died November 20, 1967.

Dr. Roy R. Kerkow of 505 Yakima Street, Wenatchee, Washington, died August 30, 1967. Dr. Kerkow was 78.

Dr. Luis Felipe Gonzales-Maldonado died on October 28, 1967, following emergency surgery for a laceration of the inferior vena cava received as a result of a stab wound sustained in his sleep at the hands of trespassers and robbers.

A native of Caguas, Puerto Rico, Dr. Gonzalez was a veteran of World War I and was a former industrial physician and chief of medical services for the Fajardo Sugar Company and Central Canovanas. He served as a physician-in-charge of the Women's Pavilion of the State Psychiatric Hospital and served also as physician for State insurance funds.

Dr. William Cleveland Miller of Gaithersburg, Maryland, died on September 24, 1967. Dr. Miller was 81.

CLASS OF 1916

Dr. Carl M. VanPoole of Mt. Airy, Maryland, died April 30, 1967, at the age of 79.

CLASS OF 1917

Dr. William B. Davidson of 42 Bruster Drive, North Kingston, Rhode Island, died on October 25, 1967.

CLASS OF 1918

Dr. Edward Joseph M. Carlin of 1423 Irving Street, Rahway, New Jersey, died July 18, 1967. Dr. Carlin was 72.

CLASS OF 1921

Dr. Fred C. Sabin, of 28 Salisbury, Little Falls, New York, died June 2, 1967, at the age of 73.

Dr. Solomon Sherman, for many years chief of the clinical laboratory at the Lutheran Hospital of Maryland, died on August 21, 1967, at his home, 2424 Eutaw Place. Dr. Sherman was 70.

A native of London, he came to Baltimore at the age of five, receiving the degree of B.S. from Loyola College in 1917 and his degree in medicine from the University of Maryland in 1921.

Following an internship and residency at Sinai Hospital, he began the practice of laboratory medicine with which he maintained a close association until his death. He was a member of the American Medical Association, the Medical and Chirurgical Faculty of Maryland, the Baltimore City Medical Society and the Southern Medical Association and the Maryland branch of the American Society of Microbiologists. For a number of years, Dr. Sherman operated a private laboratory on Reisterstown Road.

Dr. Fred B. Smith, noted Baltimore pediatrician and former chief of pediatrics at the Mercy and South Baltimore General Hospitals, died on January 29, 1968. Dr. Smith was 70 years old.

A native of Westminster, Maryland, and a graduate of St. John's College, Dr. Smith served his internship at the Maryland General Hospital, ultimately specializing in pediatrics.

Throughout his forty-seven-year career, Dr. Smith maintained a keen interest in the lives of his patients and in their families, asserting that the home, religion and education (in that order) were the most important factors in the growth of a child. His positive approach to even the most serious problem, his kindliness, thoroughness and consideration earned him the respect of hundreds of young physicians who were to benefit from his philosophy, his ability and his highly ethical approach to clinical practice.

Aside from his duties at the Mercy Hospital, Dr. Smith was active on the staffs of Union Memorial, South Baltimore General, St. Joseph's, Johns Hopkins, Maryland General and the Greater Baltimore Medical Center.

Shortly after his death, a tribute from one of his former patients, Joan S. Howe, read in part:

"Dr. Smith dispensed his wise advice and sound doctoring to my family when we were children and has been caring for my own children as well. He was always there when we needed him, regardless of time, date, weather or general inconvenience.

"We, his patients and friends, have suffered a great loss in his passing; but then, those who never met him have suffered an even greater loss, for they have missed knowing a truly great man."

CLASS OF 1922

Dr. C. Glen McCoy, of 555 Burleigh Avenue, Holly Hill, Florida, died June 15, 1967, aged 72.

Dr. Arthur Cecil Monninger of 1543 Northern Parkway died on September 29, 1967.

A general practitioner in the northern area of the city for more than 35 years, Dr. Monninger also specialized in dermatology.

A native of Scranton, Pennsylvania, he interned at the Montclair, New Jersey, Hospital and later at the St. Joseph's Hospital in Baltimore. Dr. Monninger was past president of the Downtown Lions Club and a number of fraternal organizations.

Dr. Thomas Norwood Wilson of 617 W. 40th Street, Baltimore, Maryland, died on October 28, at the age of 68.

An alumnus of St. John's College, Dr. Wilson served his internship at the University Hospital where he was one of the first residents in obstetrics following the appointment of the late Dr. Louis H. Douglas. After periods of additional training at the Sinai and St. Joseph's Hospitals, Dr. Wilson entered the practice of surgery and for a number of years was active on the staff of the Lutheran Hospital, the St.

Joseph's and Mercy Hospitals. Dr. Wilson had a private practice in Hampden and Roland Park for more than 40 years with offices located on W. 40th Street.

CLASS OF 1927

Dr. Francis (Frank) K. Morris, for many years Secretary-Treasurer of the Board of Medical Examiners of the State of Maryland, died November 8, apparently as the result of a heart attack.

A native of Baltimore and an alumnus of the St. Pius School, Loyola High School and Loyola College, Dr. Morris interned at the Union Memorial Hospital following his graduation and subsequently became resident in obstetrics and gynecology during the years 1928 and 1929. During World War II, Dr. Morris served as a senior medical officer with the Navy in Okinawa being discharged with the rank of commander.

Since 1951 he had served as an assistant professor of obstetrics and gynecology at the School of Medicine and served as chief of gynecology at the Mercy Hospital from 1948 until 1965. He also held a similar post at the South Baltimore General Hospital.

A former president of the Medical Alumni Association, Dr. Morris was also active as a staff member of the Greater Baltimore Medical Center, Union Memorial Hospital and the Bon Secours Hospital. In 1955 he was elected to the Board of Medical Examiners and served as its vice president and later as its secretary, a position he held at the time of his death. Dr. Morris was a member of the American Medical Association, a Fellow of the American College of Surgeons, a Fellow of the Southeast Surgical Conference and a diplomat of the American Board of Obstetrics and Gynecology. He was also a member of the Association of American Physicians and Surgeons.

CLASS OF 1927

Dr. Clyde Filmore Karns, of 101 W. Read Street, Baltimore, Maryland, died August 17, 1967. Dr. Karns was 66.

A native of Cumberland, Maryland, and an Alumnus of St. John's College in Annapolis, Dr. Karns served his internship and residency in surgery at the University Hospital. For a number of years he was active on the staff of the Franklin Square, Maryland General and Lutheran Hospitals. He served as consulting surgeon for the District Training School (now the Children's Center) in Laurel, Maryland, and served as surgeon in chief of the Maryland State Penitentiary and the Maryland House of Correction at Jessup, Maryland.

CLASS OF 1928

Dr. Victor Goldberg of 1916 E. 30th Street, Baltimore, Maryland, died September 18, 1967, at the age of 63.

Dr. Nathan Hersh Kotch of 1740 Ocean Avenue, Brooklyn, New York, died February 29, 1968.

Dr. Morris Lerner of 267 Macon Street, Brooklyn, New York, died September 25, 1965.

CLASS OF 1929

Dr. Henry D. Bongiorno of 516 River Street, Paterson, New Jersey, died December 4, 1967. Dr. Bongiorno was 63.

Dr. Lee Joseph Volenick of 220 Stoneyford Road, Baltimore, Maryland, died September 11, 1967. Dr. Volenick, who was 65, died in the Bon Secours Hospital in Baltimore after an illness of about six weeks.

A native of Brooklyn, New York, and an alumnus of New York University, Dr. Volenick served his internship at the South Baltimore General Hospital and later opened his practice at 4710 Liberty Heights Avenue where he continued to practice until his death. During World War II, he served as medical examiner for the Selective Service System and received letters of commendation from Presidents Roosevelt, Truman, Eisenhower, and Johnson for his continued and devoted service. He also acted as physician for the Maryland Jockey Club at both Pimlico and Timonium race tracks for more than 25 years. In recent years he

served on the Executive Staff of Bon Secours Hospital. He was a life member of the Southern Medical Association and an active member of the Medical and Chirurgical Faculty of Maryland.

CLASS OF 1931

Dr. Eugene Irving Baumgartner of 226 E. Alder Street, Oakland, Maryland, died August 8, 1967. Dr. Baumgartner was 62.

Long active in the American Medical Association and the American Academy of General Practice, Dr. Baumgartner was one of the state leaders who organized the local Maryland Academy of General Practice and for many years was active on the National Committees of the American Medical Association dealing with general practice. Dr. Baumgartner also served as an official of the Medical and Chirurgical Faculty of the State of Maryland and held numerous offices in the American Academy of General Practice.

Dr. Arthur F. Jones, chief of the Garrett County Health Department, died at his home in Oakland, Maryland, on September 23, 1967.

Dr. Harold C. Whims of the County Health Department, Asheboro, North Carolina, died July 5, 1967. Dr. Whims was 60.

Dr. Waldo Briggs Moyers of 3503 Perry Street, Mount Rainier, Maryland, died on December 9, 1967, at the age of 67. After completing his internship at University Hospital in Baltimore, Dr. Moyers began his long medical career in Mount Rainier, where he served from 1956 to 1959 as chief of medicine at Prince Georges General Hospital. He was later made a member of the hospital's board of directors. Dr. Moyers also taught for many years at Georgetown University, served for fifteen years on the board of the county Tuberculosis Association and was president of the Prince Georges County Medical Society and of the county's Heart Association. During World War II he was chairman of the Selective Service Medcal Examining Boards for both Maryland and Prince Georges County.

Dr. Moyers held important positions with the Maryland State Planning Committee, the Maryland Medical Care Committee, the Medical and Chirurgical Society of Maryland and the Prince Georges Poliomyelitis Foundation. He was a fellow of the American Geriatric Society and the American College of Cardiology.

CLASS OF 1932

Dr. William Joseph McGovern of Ocala, Florida, died on August 16, 1967, at the age of 62. A former president of the Marion County Medical Society, Dr. McGovern served on the staff of Munroe Memorial Hospital.

CLASS OF 1933

Dr. Louten Rhodes Hedgpeth of Lumberton, North Carolina, died on October 10, 1967, at the age of 59.

Dr. Frank Olaf Wolbert of 200 N. Union Avenue, Havre de Grace, Maryland, met a violent death apparently from strangulation on August 5, 1967. Dr. Wolbert was 63.

A native of Greensburg, Pennsylvania, he attended Johns Hopkins University, receiving a B.S. degree from the University of Florida. Following his graduation from the School of Medicine, he interned and completed a residency in the Harrisburg General Hospital before moving to Havre de Grace. Dr. Wolbert was chief medical examiner for Harford County selective service during World War II.

CLASS OF 1935

Dr. Aaron L. Kaminsky of Washington, D. C., died on November 3, 1967.

CLASS OF 1936

Dr. Joseph Drozd of 240 South Ann Street, Baltimore, Maryland, died November 26, 1967. Dr. Drozd was 55.

CLASS OF 1937

Dr. William C. Gordon, of 648 Ringwood

Avenue, Wanaque, New Jersey, died May 29, 1967. Dr. Gordon was 56.

Dr. Eldridge H. Wolff of Cambridge, Maryland, died on December 7, 1967, at the age of 59. Following internships at Baltimore City Hospitals and with the medical department of the Maryland House of Correction, Dr. Wolff worked in obstetrics at University Hospital and in general medicine at the Johns Hopkins University School of Medicine. In addition to serving two terms as chief of staff at Cambridge-Maryland Hospital, he was president of the Dorchester County Medical Society and of the Dorchester County Tuberculosis and Health Association. Dr. Wolff was a member and fellow of the American Medical Association and a member of the Medical and Chirurgical Faculty of Maryland, the Physicians Review Board and Maryland Hospital Service, Inc., of Baltimore.

CLASS OF 1940

Dr. James R. Dwyer, a surgeon and staff member of the Washington County Hospital, died July 8, 1967, following a heart attack while on vacation at Ocean City, Maryland.

A native of Renovo, Pennsylvania, and an alumnus of the Mount St. Mary's College in Emmitsburg, Maryland, Dr. Dwyer served his internship and residency at the St. Agnes Hospital from 1940 until 1944. After a period of active duty with the United States Navy, he then moved to Hagerstown where he practiced surgery. He was a member of the American College of Surgeons, the International College of Surgeons and the Medical and Chirurgical Faculty of Maryland. He was also on the review board of the Maryland Hospital Service, on the Board of Directors of the Washington County Agricultural and Mechanical Association. Dr. Dwyer is survived by his parents, Dr. and Mrs. Frank P. Dwyer of Renovo, Pennsylvania, and a brother, Dr. Frank P. Dwyer, Jr. of Baltimore.

CLASS OF 1943

Dr. John W. Epperson of 968 North East 40th Street, Boca Raton, Florida, died November 17, 1967. Dr. Epperson was 48.

CLASS OF 1944

Dr. Perry Futterman of Beckley, West Virginia, died on November 30, 1967, at the age of 46. After an internship with University Hospital in Baltimore, Dr. Futterman served as chief of the Chest Disease Service at the United States Naval Hospital in Oak Knoll, California, then headed the emergency room at the United States Naval Dispensary in Yokosuka, Japan. Following a two-year residency at the Boston City Hospital, he was awarded a fellowship for research in diabetes, endocrine and metabolic problems with the May Institute in Cincinnati. Dr. Futterman also served in the Korean War, after which he was with the departments of medicine and clinical pathology at the University of Maryland School of Medicine.

Since 1956 Dr. Futterman has been a specialist in internal medicine at the Beckley Appalachian Regional Hospital, of which he became chief of staff in June, 1967. He was a member of the American Medical Association, the American Association of Military Surgeons, the American Diabetes Association, the American Geriatrics Society, the Raleigh County Association for Mental Health and the Raleigh County Cancer Association.

CLASS OF 1953

Dr. Wyland F. Doerner, Jr. of 414 N. Mechanic Street, Cumberland, Maryland, died recently.

Dr. Frederick A. Garlock of 1530 Dover No. 3, Denver, Colorado, died recently.

BULLETIN *School of Medicine*
University of Maryland

VOLUME 53 JULY, 1968 NUMBER 3

TABLE OF CONTENTS

the more frequent her migraine...
the more severe her attacks...
the more the need for Sansert

the only known specific for the prophylaxis of migraine attacks

Sansert, whose action is preventive, is for severe migraine sufferers: (1) the patient who experiences one or more attacks a week; (2) the patient whose episodes are so overwhelming and refractory to symptomatic treatment that preventive therapy is indicated regardless of frequency.

With this unique prophylactic drug, the patient may not only gain freedom from many such attacks—perhaps even complete freedom from migraine—but may also, as a result, enjoy release from the constant fear of recurrent incapacitation.

It is important that the physician be familiar with the supervisory procedures required in the administration of the drug in order to minimize the possibility of adverse effects, particularly retroperitoneal fibrosis.

Contraindications: Pregnancy, peripheral vascular disease, severe arteriosclerosis, severe hypertension, coronary artery disease, phlebitis or cellulitis of the lower limbs, pulmonary or collagen disease, impaired liver or renal function, cachectic or septic states.

Warnings: Retroperitoneal fibrosis, a related condition (pleuropulmonary fibrosis), and cardiac murmurs or vascular bruits have been noted. Warn patients and caution them to report immediately cold, numb and painful hands or feet, leg spasms on walking, any type of chest, girdle or flank pain or other untoward symptoms. If symptoms develop, discontinue drug.

Interrupt therapy for 3-4 weeks every six months.

Precautions: Patients should remain under constant supervision and be examined regularly for fibrotic or vascular complications. (See **Adverse Reactions.**)

Surgical intervention can often be avoided since fibrotic manifestations and vascular shutdown have usually regressed upon withdrawal of drug. Cardiac murmurs have sometimes persisted.

Sansert is indicated for prophylactic treatment of vascular headache, not for the acute attack.

Adverse Reactions: 1) Fibrotic Complications—Retroperitoneal fibrosis may present clinically with general malaise, fatigue, weight loss, backache, low grade fever (elevated sedimentation rate), urinary obstruction (girdle or flank pain, dysuria, polyuria, oliguria, elevated BUN), vascular insufficiency of the lower limbs (leg pain, Leriche syndrome, edema of legs, thrombophlebitis). Pleuropulmonary fibrotic complications usually present clinically with dyspnea, tightness and pain in the chest, pleural friction rubs, and pleural effusion. Cardiac fibrotic complications usually present clinically with cardiac murmurs and dyspnea. One case of fibrotic plaques simulating Peyronie's disease. 2) Vascular Complications—Retroperitoneal fibrosis may result in vascular insufficiency of the lower limbs. Intrinsic vasoconstriction of large and small arteries may present with chest pain, abdominal pain, or cold, numb, painful extremities with diminished or absent pulses. Ischemic tissue damage is rare. 3) Gastrointestinal Symptoms—Nausea, vomiting, diarrhea, heartburn, abdominal pain. 4) CNS Symptoms—Insomnia, drowsiness, mild euphoria, dizziness, lightheadedness, hyperesthesia, unworldly feelings (described variously as "dissociation," "hallucinatory experiences," etc.). 5) Dermatological Manifestations—Nonspecific rashes, increased hair loss. 6) Edema. 7) Weight Gain

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University of Maryland

VOLUME 53 JULY, 1968 NUMBER 3

Editorial

DEAN STONE RETIRES

DR. WILLIAM S. STONE, Dean of the School of Medicine, formally retired June 30, 1968. Dr. Stone's tenure as Dean encompassed more than a decade and a half during which time the physical appearance, the internal organization, and the motivation of the School of Medicine experienced a significant and important change. New departments have been added. Great expansion of basic research has been achieved. Many full-time faculty positions have been created and extensive changes have been undertaken and completed in the structure of the undergraduate curriculum. These changes have included certain elements of leadership as well as the school's reaction to national and worldwide trends in medical education and research.

At this hour, and in its 161st year, the School happily finds itself in a pivotal position, adequately staffed, and equipped to react progressively and properly to the increasing demands of medical science, research, and education in a rapidly evolving scientific environment.

High Dosage Sodium Colistimethate Therapy

STUART H. WALKER, M.D., PERRY SHELTON, M.D., ROBERT R. HOLTHUS, M.D.

Introduction

The therapeutic use and efficiency of sodium colistimethate is limited by its toxicity and particularly by its nephrotoxicity.[3, 5] Its antibacterial effectiveness and toxicity are both directly related to dosage.[4] The intended injury to bacterial cell membranes and the complicating toxic injury to mammalian cells seem to be consequent to the same mechanism.[4] Many Gram-negative organisms are not sensitive to the serum and tissue concentrations achieved at the usually recommended dosage, 2.5 and 5.0 mg. per kilogram of body weight per day, nor to those achieved at 10 mg. per kilogram per day.[6, 9]. Optimal dosage, therefore, should be higher and probably should be the highest which can be tolerated without significant host cell injury. Previous studies have demonstrated that infants and children are less susceptible than adults to host cell injury at equivalent dosage schedules based upon weight.[8] The present study was intended to determine the maximum dosage of sodium colistimethate which could be safely tolerated by infants and children at various ages.

Abstract

Sixty-two infants and children with suspected or proven Gram-negative enteric bacterial infections were treated with intramuscular sodium colistimethate. Dosage in 52 patients was 20 mg. per kilogram per day, while in 10 it was 10 mg. per kilogram per day. Patients were evaluated to determine the effectiveness of therapy, the drug serum levels achieved and the presence of alterations in renal functions. There were 7 deaths, 3 consequent to detected Gram-negative bacterial infection. All other patients recovered from their infectious disease without residua of the disease or of drug toxicity. Transient cylindruria or azotemia occurred in 22 patients and were directly both dose and age related. Four older children demonstrated 1 to 4 day periods of marked oliguria. Recovery of renal function was complete and rapid in all surviving patients. Serum levels of sodium colistimethate were directly related to dose, were greatest between 30 minutes and 1 hour after dosing, 6 to 18 mcg. per milliliter at a dosage of 20 mg. per kilogram per day, and were always less than 2 mcg. per milliliter after 6 hours. Although all observed nephrotoxicity was reversible, dosage should be adjusted to age to obviate the occurrence of transient oliguria. Dosage of 20 mg. per kilogram per day, well tolerated in newborns and young infants, should be reduced with advancing age to 10 mg. per kilogram per day in older children and further reduced or eliminated in the presence of decreased renal output.

Material and Methods

The case material utilized in this study were nursery and pediatric patients admitted to the Pediatric Service, Mercy Hospital, Baltimore, Maryland, during the period 1 July, 1965 to 1 July, 1967. All newborns, infants and children with known or suspected major infections due to Gram-negative enteric bacilli observed during this period were admitted to the study. Twenty-seven patients were subsequently determined to have specific infection by isolation of Gram-negative enteric organisms from cultures of blood, cerebrospinal fluid, urine, or needle aspirates. Eighteen patients were determined to be diseased and an infection with a Gram-negative enteric bacillus was presumed, but no etiologic organism was directly isolated. Seventeen patients were

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treated because of the likelihood of an infection with the Gram-negative enteric bacillus, but no infection was demonstrated. All patients received sodium colistimethate (Colymycin Injectable, without buffer or dibucaine, supplied by Warner-Chilcott Laboratories) intramuscularly in equally divided doses at 6 hour intervals or intravenously by continuous drip. All of the newborns and 26 of the older infants and children received 20 mg. per kilogram per day while 10 of the older infants and children received 10 mg. per kilogram per day. Additional therapy was provided as indicated and did not vary significantly from usual management procedures. Additional antibiotics were not utilized. A few treated patients who were determined to have infections with Gram-positive organisms were subsequently treated with other antibiotics and excluded from the study.

Prior to the initiation of therapy, material for bacteriologic isolation was obtained from the blood in all patients and from the local secretions, stool, urine and/or cerebrospinal fluid as indicated if local infection was suspected or present. A direct lung aspiration with a 20-gauge needle attached to a syringe was utilized to obtain material for culture in all patients with pneumonia who were admitted to the study. In addition to other appropriate examinations a complete blood count, urinalysis, and blood urea nitrogen determination were obtained in each prior to the initiation of therapy and at least every 2nd day thereafter until the completion of therapy. If abnormalities were detected, these studies were repeated after the completion of therapy until they had returned to normal. Cultures of local secretions were repeated every second day until after the completion of therapy, and cultures of blood were repeated at least every 4th day in patients suspected of bacteremia. Creatinine clearance was measured immediately after recovery and 6 months to 1 year later in those patients who demonstrated significant nephrotoxicity. Sodium excretion rates were determined during episodes of oliguria and after recovery in those patients who demonstrated oliguria. All isolated Gram-negative

Proven Infection—28	Eradicated	Improved	Failure
Sepsis—E. coli	1		
Pseudomonas	1		1* (burn)
D. Pneumoniae			1*
Meningitis—E. coli		1	1*
A. aerogenes	1		1*
Pneumonia—E. coli	1		
H. influenzae	1		
Pseudomonas	1		
Peritonitis—Pseudomonas	1		
Omphalitis—E. coli	1		
Mastoiditis—Pseudomonas	1		
Abscess—Pseudomonas	2		
A. aerogenes	1		
Enteritis—Shigella flexneri	1		
Urinary Tract Infection	9		1
Disease—Etiology Undetermined—17	Eradicated	Improved	Failure
Pneumonia	7		
Peritonitis	2		1*
Meningitis	3		
Osteomyelitis	1		
Wound Infection	2		
D-Trisomy with sepsis (possible)			1*
Prophylaxis—17			
Neonatal sepsis		13	
Miscellaneous		4	
TOTAL—62	37	18	7

* DEATH

Table 1 THERAPEUTIC EFFECT OF SODIUM COLISTHEMETHATE IN 62 INFANTS AND CHILDREN

enteric bacilli were demonstrated to be sensitive to sodium colistimethate by the single disc-agar method (inhibition of growth surrounding a 10 mcg. disc).

Therapy was continued in the initial dosage for a minimum of 5 days and until the patient was considered completely recovered. In 4 instances it was necessary to discontinue therapy at an earlier time because of the appearance of oliguria.

The sodium colistimethate content of the serum of all treated patients was assayed at various times, usually 24 and 72 hours after the initiation of therapy. Serum specimens were obtained at various intervals after the most recent dose to determine the persistence of the serum concentrations. All assays were performed by the Waldemar Medical Research Foundation using the cylinder plate bio-assay method and Bordetella bronchispectica ATCC4617 as the test organism. The zone of inhibition produced by the sera containing sodium colistimethate is compared to that produced by known concentrations of colistin base and the bio-assay reported in "base equivalents" (as micrograms of colistin base effect per milliliter of serum).

Results

Amongst 27 patients with proven infection due to Gram-negative bacilli, therapy resulted in cure in all but 4 (Table 1). Only

AGE	NEWBORNS	INFANTS 1-12 months		CHILDREN 1-5 yrs		CHILDREN 5-13 yrs		TOTAL
SODIUM COLISTHEMETHATE DOSAGE (mg/kg/24 hrs)	20	20	10	20	10	20	10	
NEPHROTOXICITY	6	3	0	2	0	8	3	22
Cylindruria	4	3	0	2	0	8	3	20
Albuminuria	4	0	0	0	0	0	0	4
Azotemia	3	0	0	2	0	6	0	11
Oliguria	0	0	0	1	0	3	0	4
NO TOXICITY	19	9	4	5	3	0	0	40
TOTAL	25	12	4	7	3	8	3	62

Table 2 Incidence of nephrotoxicity associated with sodium colisthemethate therapy related to age and dose

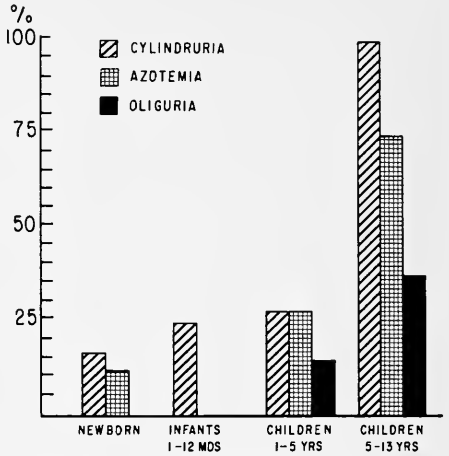

Fig 1 PERCENTAGE OF VARIOUSLY AGED PATIENTS DEMONSTRATING NEPHROTOXICITY WHILE RECEIVING SODIUM COLISTIMETHATE 20 MG/KG/24 HRS

one of the 4 patients with meningitis due to a Gram-negative bacillus survived. One patient, with hydrocephalus and E.coli meningitis, was considered to have been improved and spinal fluid was sterile before death. One patient with E.coli and one patient with A.aerogenes meningitis died within 9 and 34 hours of the commencement of therapy. The other failure in this group was a patient who died with extensive burn wounds and Pseudomonas sepsis. One patient died with a fulminant sepsis proved after death to be pneumococcal. Two of 18 patients with infectious disease of initially undertermined etiology died, one with meconium peritonitis and one with D-trisomy and probable sepsis. Seventeen patients who were treated expectantly because of a high risk of Gram-negative bacillary infection recovered without evident infection.

Nephrotoxicity evidenced by cylindruria, usually coarse granular casts, was detected in 22 of the 62 treated patients. Azotemia evidenced by a rise in blood urea nitrogen concentration greater than 10 mg per 100 milliliter was detected in 11 patients. Significant oliguria was detected in 4 patients.

The incidence of nephrotoxicity increased with age. Oliguria was noted in 1 two-year-old patient and in 3 ten-year-olds treated at a dosage of 20 mg. per kilogram per day. This dosage was tolerated with only minimal cylindruria in 4 and transient azotemia in 3 of 25 newborns (Table 2 and Figure 1). All evidences of nephrotoxicity were observed to clear completely after cessation of therapy.

In each of the 4 patients who demonstrated oliguria a depression of renal excretion may have been associated with the disease itself, its consequent dehydration, or endotoxemia. A gradual reduction in urinary volume and a gradual rise in blood urea nitrogen preceded the appearance of oliguria in 3 instances (Figures 2, 3 4). Despite the cessation of colistimethate therapy, oliguria, once initiated, persisted for approximately 3 days in each of the 3 surviving patients in association with a rapid resolution of their infectious disease. Diuresis appeared thereafter and a return to normal urine volume was evident within a week in each patient. Blood urea nitrogen elevation and changes in the urine sediment returned to normal within 2 weeks. Sodium excretion was measured in 3 of the oliguric patients and varied little from normal during either oliguria or diuresis. A 4th patient demonstrated oliguria from the time of initiation of therapy until death 36 hours after admission from what was subsequently determined to be pneumococcal sepsis.

Patients with oliguria were reevaluated between 6 months and 1 year after treatment to detect persistent evidence of nephrotoxicity. Blood urea nitrogen concentration, urinalysis, concentrating ability and creatinine clearance were all normal at this time.

No evidence of neurotoxicity, neuromuscular blockade or other toxicity was detected in any patient.

Assays of the serum concentration of

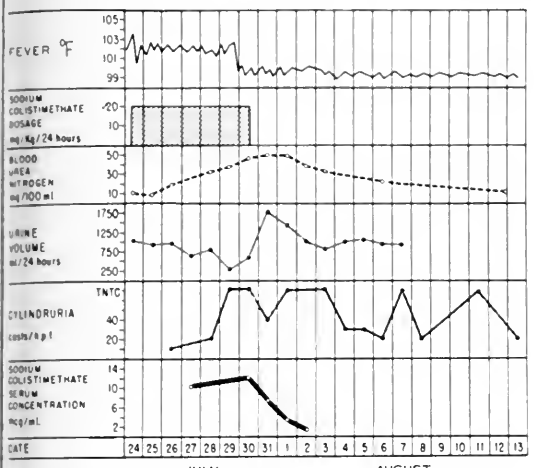

Figure 2 TC COURSE OF SODIUM COLISTIMETHATE ENTERITIS WITH OLIGURIA

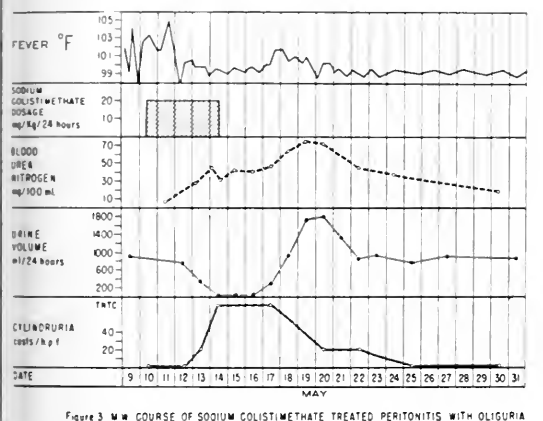

Figure 3 MW COURSE OF SODIUM COLISTIMETHATE TREATED PERITONITIS WITH OLIGURIA

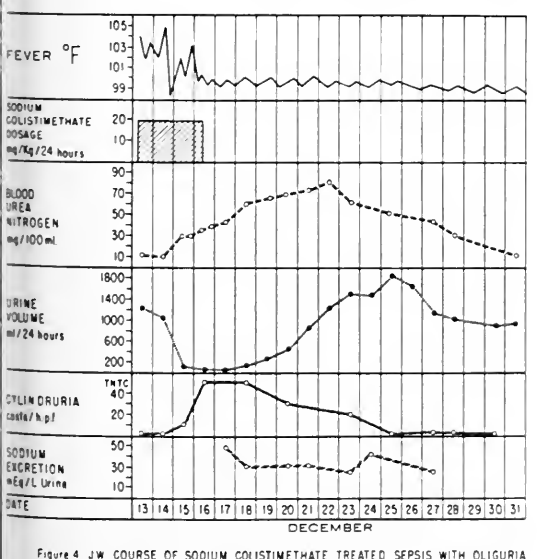

Figure 4 JW COURSE OF SODIUM COLISTIMETHATE TREATED SEPSIS WITH OLIGURIA

sodium colistimethate in infants and older children at varying intervals after intramuscular dosing at 20 mg. per kilogram per day revealed no apparent age-determined difference (Figures 5, 6, 7). Maximum concentrations were achieved in the first hour and were between 6 and 18 mcg. per milliliter. Concentrations were regularly less than 3 mcg. per milliliter 6 hours after dosing and no evidence of accumulation was evident after multiple doses. The first half life of serum concentration in newborns was 1.4 hours, in infants 1-12 months of age, 1.7 hours and in children 1-13 years of age, 1.4 hours.

Discussion

Serum concentrations above 10 mcg. per milliliter during the 1st hour after dosing and concentrations between 2 and 10 mcg. per milliliter throughout therapy were regularly achieved in newborns, infants and children treated with sodium colistimethate at 20 mg. per kilogram per day. Dosage at 5.0 mg. per kilogram per day produces maximum serum concentrations between 3 and 5 mcg. per milliliter and dosage at 10 mg. per kilogram per day produces maximum serum concentrations between 4 and 7 mcg. per milliliter.[8] The higher dosage seems to result in a significant gain in serum and presumably tissue concentration of the drug.

In vitro sensitivity studies have indicated that sodium colistimethate concentrations of 10 mcg. per milliliter are bactericidal in only 80% of E.coli strains, 93% of Pseudomonas strains and 80% of Klebsiella strains.[6] Dosage approximating 20 mg. per kilogram per day is required to regularly achieve this concentration in children.

Enhanced serum concentrations are obtained at the cost of an increased incidence of nephrotoxicity in older children, but not in newborns or infants less than one year of age. Treatment schedules of 5.0 and 10 mg. per kilogram per day have been associated with transient azotemia in 14% of young infants[8] and in this study it appeared in 12%. Oliguria was not noted in any treated patient less than 2 years of age and the

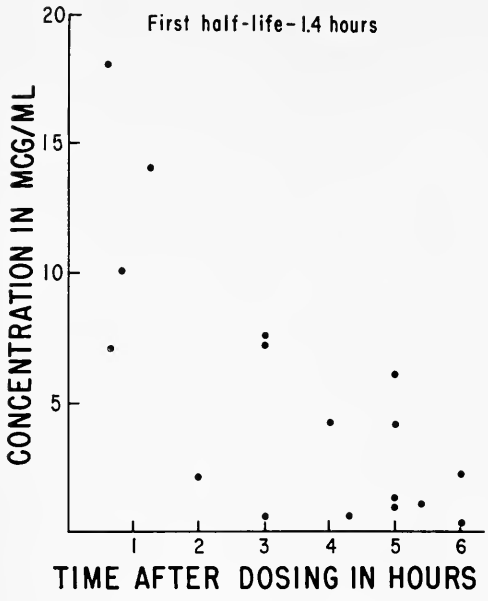

Figure 5 SERUM CONCENTRATION OF SODIUM COLISTIMETHATE IN NEWBORNS AFTER DOSING AT 20 MG/KG/24 HOURS

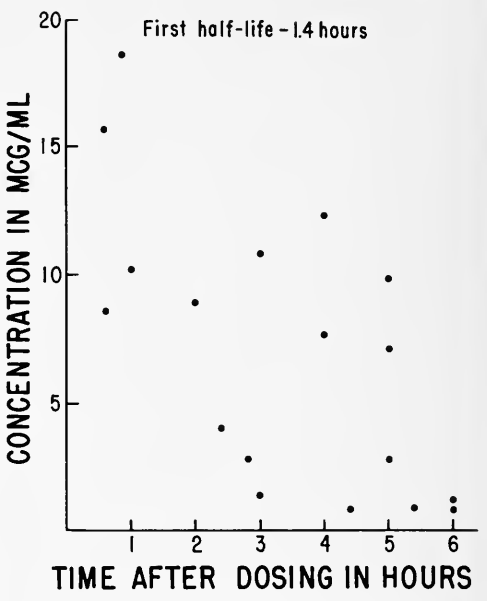

Figure 7. SERUM CONCENTRATION OF SODIUM COLISTIMETHATE IN CHILDREN (1-13 YEARS) AFTER DOSING AT 20 MG/KG/24 HRS

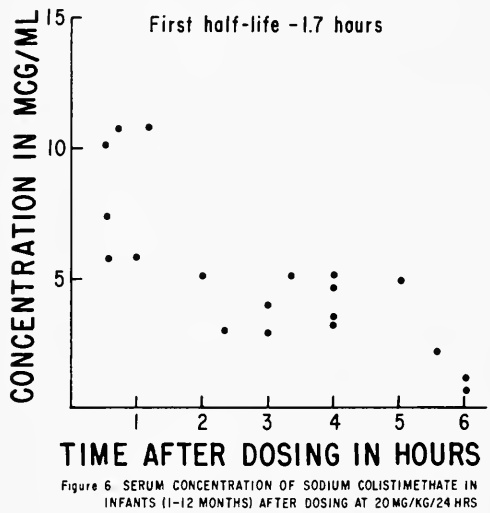

Figure 6 SERUM CONCENTRATION OF SODIUM COLISTIMETHATE IN INFANTS (1-12 MONTHS) AFTER DOSING AT 20 MG/KG/24 HRS

infrequent cylindruria and azotemia cleared within 2 days of the cessation of colistimethate therapy in each instance.

No difference between the incidence of nephrotoxicity in infants and in older children was evident in a previous study of sodium colistimethate therapy using 5 and 10 mg. per kilogram per day.[8] At 20 mg. per kilogram per day, however, older children demonstrated a greater incidence of nephrotoxicity than did infants. There is no evidence from this study or the study of Axline, *et al.*[1] that the excretion rate of sodium colistimethate in prematures, full term newborns, or young infants is greater than in older children. The serum concentrations achieved by dosage calculated on the basis of body weight and the serum half life of the drug's antibiotic effect, as indicated by bio-assay against a test organism, are not significantly different at these different ages. If antibiotic effect is a measure of cell membrane toxicity, then the toxic effect of similar antibiotic concentrations and similar durations of concentration should be equivalent. However, inasmuch as similarly effective antibiotic concentrations produce a different incidence and severity of nephrotoxicity, a difference in tissue response must be postulated. It seems reasonable to presume that the renal tubular cells of infants are more resistant than those of older children and adults to the toxic effects of sodium colistimethate.

Administration of sodium colistimethate and other polymyxins at high dosage to rats and mice results in renal medullary

and cortical hyperemia.[4, 7] Rats which are permitted to survive, however, demonstrate a return to normal of the appearance of the renal parenchyma.[4] Percutaneous biopsies in patients receiving sodium colistimethate have failed to reveal any histological evidence of nephrotoxicity.[2, 9] In the present study all patients who demonstrated nephrotoxicity including the older children with oliguria, cylindruria and azotemia had complete recovery of renal function. It seems reasonable to conclude that even nephrotoxicity manifested by marked oliguria is completely reversible.

In the patients who demonstrated oliguria in the present study a gradual reduction in urine flow preceded an abrupt reduction in volume and an abrupt rise in blood urea nitrogen concentration. Careful monitoring of urine volume when older children are treated with high doses of sodium colistimethate should permit early detection of incipient oliguria, cessation of therapy and either prevention or amelioration of the oliguric period. Although the oliguric episode is unassociated with any persisting tissue injury, its management is fraught with sufficient, usually iatrogenic, hazards that it should be avoided. Dosage should be modified accordingly and, inversely proportional to age, be adjusted downwards from 20 mg. per kilogram per day in newborns and infants.

Conclusions

Sodium colistimethate administered intramuscularly in a dosage of 20 mg. per kilogram of body weight per day is well tolerated in newborns and young infants. Significant though reversible nephrotoxicity may result from this dosage in older children. Dosage should therefore be reduced with advancing age to 10 mg. per kilogram per day in children over 2 years of age and further reduced or eliminated in the presence of decreased renal output.

Acknowledgments

The assistance of Dr. Victoria Maisog in the organization of the study and the preparation of the manuscript is gratefully acknowledged.

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The Goodpasture Syndrome*

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In recent years, increasing attention has been focused on a syndrome characterized by pulmonary hemorrhage and glomerulonephritis. Osler, in 1895, noted hemoptysis and nephritis in two of eleven cases of erythema exudativum multiform.[1] At the time of the influenza pandemic of 1918-19, Goodpasture described a young man with recurrent hemoptysis, anemia and proteinuria who was found at autopsy to have intra-alveolar hemorrhage and glomerulonephritis.[2] Between 1919 and 1955 only scant reports of similar cases were reported in the literature.[3,4,5] The term "Goodpasture's syndrome" was popularized by Stanton and Tange in 1958.[6] Since that time over 100 cases have been recorded in the world literature. The purpose of this paper is to present four additional cases of Goodpasture's syndrome, one of which is the first known occurrence in a Negro, and to bring special attention to the distinct pathological features of the syndrome.

Case Reports

Case 1, E. J. (U. H. 69-940). A 26-year-old female developed a cough one year prior to admission. Three months later hemoptysis appeared. Tea-colored urine was intermittently noted for six months. Shortly prior to admission she suffered a miscarriage followed by progressive dyspnea and weakness.

Upon admission, physical examination revealed a pale white female with a blood pressure of 120/70 mm. of Hg., a pulse rate of 90 per minute and labored respirations at 26 per minute. There were diffuse rhonchi audible over both lung fields. The remainder of the physical examination was normal. (See Table 1 for laboratory information.) A chest roentgenogram revealed a soft infiltrate in the lower half of both lung fields with greatest involvement at the bases. She was treated for apparent pulmonary edema. Her hospital course was one of rapid deterioration characterized by anemia, hemoptysis, tachypnea, cyanosis and acute renal failure. She expired on her twelfth hospital day. The duration of her illness was nine months.

Autopsy findings: The peritoneal cavity contained two liters of serous fluid. The heart weighed 300 Gm. and the lungs 2050 Gm. The majority of alveoli in sections from all lobes were filled with red cells. There was extensive exudation of fibrin and hemosiderin-pigmented macrophages. Fibrous organization was seen in scattered alveoli. The alveolar walls were not thickened.

The kidneys weighed 200 Gm. each. The subcapsular surfaces were smooth with numerous petechiae. All the glomeruli showed crescents, mostly composed of epithelial cells (Fig. 1). Hyaline droplets were present in epithelial cells in a moderate number of glomeruli. There were frequent spaces or pseudotubules within the epithelial crescents. Cellular proliferation in the tufts was moderate or absent. The glomerular tufts were ischemic, with wrinkled basement membranes and fibrillar deposits of P.A.S. staining material. The renal tubules showed extensive but patchy atrophy, with diffuse interstitial fibrosis and mild lymphocytic infiltration in the cortex. Red cell, granular hemoglobin and hyaline casts were numerous. There was mild arterial intimal fibrosis.

Case 2, T. S. (U. H. 20-85-59). A 36-year-old white male developed anemia, weakness, joint swelling and arthralgia five years and ten months prior to admission. Medical and hematological evaluation at that time were non-diagnostic. Microscopic hematuria was

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Table I Laboratory Data

Case	Hemoglobin g./100 cc.	Hematocrit %	Leucocytes cu. mm.	L-E cell	Blood Urea Nitrogen mg/100 cc.	Albumin g./100 cc.	Globulin g./100 cc.
I	7.8	22	11,800	negative	44 to 266	2.7	2.0
II	5.2	18	16,650	negative	12 to 192	3.8	2.2
III	4.1	13.5	18,200	negative	24 to 136	3.3	2.3
IV	3.7	13	8,900	negative	25 to 96	3.0	2.4

Bleeding time, clotting time, prothrombin time, platelets and Rumple Leeds test were normal in all cases.

Urinalysis

Case	Ph	Protein	Protein g./24 hr.	Glucose	Leucocytes HPF	Erythrocytes HPF	Casts Red	Casts White	Casts Granular
I	Acid	heavy	unknown	negative	0–5	20–40	2–4	0	2–4
II	Acid	heavy	unknown	negative	2–3	20–30	0	0	0
III	Acid	heavy	3.0	negative	2–3	15–20	0	0	2–3
IV	Acid	heavy	5.8	negative	1–2	40–60	1–2	0	1–2

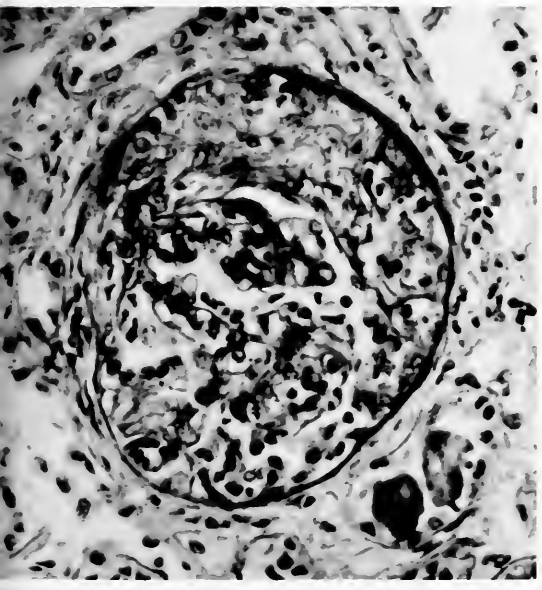

Figure 1. Case 1. Glomerular epithelial crescent. The glomerular tuft appears nearly normal except for extensive adhesion to Bowman's capsule. P.A.S. stain X 360.

noted four years prior to admission and scant hemoptysis had occurred intermittently for three years. Azotemia was initially noted four months prior to admission. Six hours before admission he became acutely dyspneic and had brisk hemoptysis.

Physical examination revealed a pale white male with a blood pressure of 170/95 mm. of Hg. The pulse rate was 128 per minute and respirations were labored at 40 per minute. He was coughing up bright red blood. Diffuse rales and rhonchi were audible over both lung fields. Cardiomegaly and mild hepatomegaly were also noted. The remainder of the physical examination was normal. Chest roentgenogram revealed a nonhomogenous infiltrate in the lower two-thirds of both lung fields. (See Table I for laboratory information.)

The patient was digitalized, tranfused and given oxygen. His course was one of increasing tachypnea and hemoptysis, with death occurring 20 hours after admission. The immediate cause of death was respiratory insufficiency, and the entire illness lasted 70 months.

Autopsy findings: Petechiae were noted in the skin and mucosa of the palate, stomach, small intestine and trachea. The heart weighed 530 Gm. and lungs 3700 Gm. There was ex-

tensive patchy alveolar hemorrhage, marked edema and alveolar exudation of hemosiderin-pigmented macrophages. Alveolar walls were diffusely thickened by swelling of alveolar lining cells, which were frequently cuboidal. Pulmonary arteries showed mild intimal fibrosis and extensive basophilic deposits on elastic fibers, staining positively for iron and with Von Kassa's stain for calcium. The kidneys together weighed 300 Gm. The subcapsular surfaces were granular. There was a marked deficiency in number of glomeruli, and most of those remaining were hyaline and scarred. Most of the unscarred glomeruli showed focal adhesion to Bowman's capsule and a few showed pseudotubule formation in Bowman's space. There was marked patchy tubular atrophy, alternating with hypertrophy of proximal convoluted tubules. Moderate fibrosis and lymphocytic infiltration was seen in interstitial tissue. There was focal arteriolar sclerosis.

Case 3, E. C. (U. H. 28-89-33). A 22-year-old Negro female was admitted to University Hospital because of abdominal pain, hemoptysis and renal failure. At six months of age she developed pulmonary tuberculosis and was hospitalized for three years. Two years before her final admission, microscopic hematuria was noted. Three months later her hemoglobin was 9.6 Gm./100 ml., and a urinalysis revealed three plus proteinuria and microscopic hematuria. Nine months prior to final admission she was hospitalized for anemia and hemoptysis. A chest roentgenogram showed a soft infiltrate in the right upper and middle lung fields. In spite of negative sputum examinations for tubercle bacilli, she was felt to have recurrent pulmonary tuberculosis. She was placed on antituberculosis therapy and transferred to a state tuberculosis hospital. Her course rapidly deteriorated. When she was placed on high doses of prednisone her general condition greatly improved and within four days hemoptysis had ceased. One month later her chest roentgenogram was normal and azotemia was first noted. An intravenous pyelogram showed poor dye concentration bilaterally. In the one month prior to admission she developed severe anemia, abdominal pain, increasing azotemia and brisk hemoptysis. She was transferred to University Hospital in a moribund state.

Upon admission, physical examination revealed an acutely and chronically ill Negro female with swollen eyelids, who was coughing up bright red blood. Her blood pressure was 160/100 mm. of Hg.; pulse rate was 160 per minute and respirations were labored at 40 per minute. Chest examination revealed diffuse bubbling rales and rhonchi. The heart was clinically enlarged and a ventricular gallop was present. There was mild hepatomegaly. The remainder of the physical examination was normal. Chest roentgenogram showed a diffuse soft infiltrate involving the major portion of the right lung and the left perihilar region. (See Table I for laboratory information.)

She was continued on antituberculous therapy and prednisone. She was digitalized and cautiously transfused. Her hospital course was characterized by marked tachypnea, cyanosis, increasing azotemia, hypertension and oliguria. She received peritoneal dialysis but expired on the tenth hospital day. The duration of her illness was 22 months.

Autopsy findings: The heart weighed 390 Gm. and the lungs 1550 Gm. There was extensive patchy bronchopneumonia. Sections of most lobes showed marked alveolar hemorrhage and exudation of hemosiderin-pigmented macrophages. Many alveoli contained fibrin masses covered by a single layer of flattened cells with plump nuclei resembling the alveolar lining. There was patchy fibrous organization. Scattered fusiform areas of fibrous tissue contained central calcified and ossified masses (Fig. 2). One medium-sized artery was

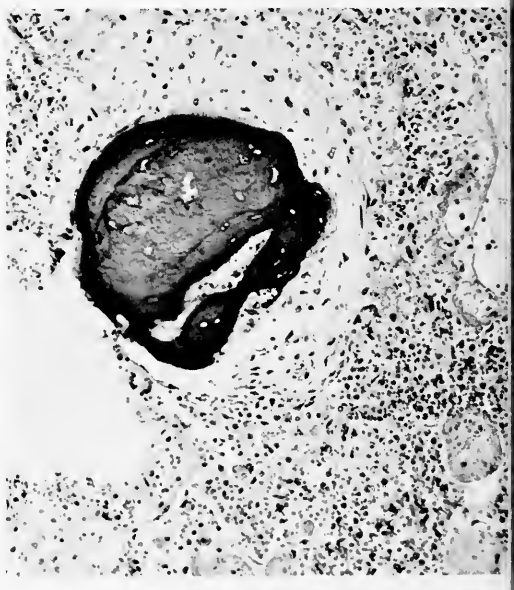

Figure 2. Case 3. Central ossification in ovoid area of fibrous tissue in lung. Recent hemorrhage and fibrin exudate in alveoli. H and E. X 200.

occluded by an organized thrombus. No evidence of healed tuberculosis was found.

Each kidney weighed 80 Gm. The subcapsular surfaces were granular. Nearly all the glomeruli were hyaline and scarred. The few unscarred glomeruli showed epithelial crescents and pseudotubule formation. The best preserved glomerular tufts were of normal cellularity, although several showed an increase in fibrillar P.A.S.-positive material. There was marked atrophy and focal dilatation of tubules, with patchy interstitial fibrosis and lymphocytic infiltration. A moderate number of hyaline and hemoglobin casts were noted. Fibrinoid degeneration and hemorrhage were seen in the walls of occasional arterioles, and there were organizing thrombi in some of the small arteries. A section of stomach showed acute and healed vasculitis in the submucosa, with fibrinoid degeneration in the wall of an arteriole and fibrous obliteration of the lumens of small arteries. There was an organized hematoma (10 x 8 cm.) below the lower pole of the right kidney.

Case 4, J. D. (U. H. 32-06-41). A 38-year-old white male developed weakness, shortness of breath and decreased vision in his right eye 3½ months prior to his admission. At the onset of symptoms he was admitted to another hospital and found to have bilateral bronchopneumonia, hematuria, proteinuria, and a hypochromic, microcytic anemia. An intravenous pyelogram showed normal size kidneys but diminished dye excretion bilaterally. He was treated with antibiotics, blood transfusions and iron and showed some clinical improvement. However, his chest roentgenogram never cleared completely. Blood urea nitrogen was 25 mg./100 ml. on admission and 56 mg./100 ml. at the time of discharge 30 days later. He remained on bed rest at home for seven days and noted that his weakness was increasing. Five days prior to his admission be became markedly short of breath and hemoptysis occurred. These symptoms persisted until the time of admission.

On admission he was a profoundly pale male in acute respiratory distress. His blood pressure was 170/80 mm. of Hg., the pulse rate was 140 per minute and respirations were labored at 50 per minute. A large right macular hemorrhage was noted. Diffuse coarse rales were audible over both lung fields. Cardiomegaly and a systolic ejection murmur were evident. The liver was greatly enlarged. The remainder of the physical examination was normal. The chest roentgenogram showed a diffuse soft infiltrate involving both lung fields, with greatest involvement on the left. (See Table I for laboratory information.) Prussian blue stain of the sputum revealed hemosiderin-laden macrophages.

He was placed on high doses of penicillin, methicillin and prednisone. He was digitalized and cautiously transfused. He became cyanotic following transfusion. A muscle biopsy was normal. His condition rapidly deteriorated and on the third hospital day his arterial oxygen saturation was 74% while receiving oxygen under positive pressure. He expired on the sixth hospital day. The cause of his death was respiratory insufficiency and his entire illness lasted 14 weeks.

Autopsy findings: The heart weighed 480 Gm. and the lungs 2650 Gm. Sections from all lobes showed marked diffuse alveolar hemorrhage, patchy fibrinous exudate and hemosiderin-pigmented macrophages. There was extensive patchy interstitial and alveolar fibrosis. Fibrin thrombi were seen in occasional alveolar capillaries. The alveolar walls and fibrous alveolar plugs were covered with a layer of large flat or round cells with deeply staining cytoplasm and large single or multiple nuclei with prominent nucleoli. Sheets and loose aggregates of similar but polygonal cells filled many alveolar spaces, intermingled with more rounded siderophages (Fig. 3). Fibrous plugs extended into some of the small bronchi.

Each kidney weighed 270 Gm. The subcapsular surfaces were smooth. There was a small, old infarct in the right lower pole. Most of the glomerular tufts were adherent to Bowman's capsule, with proliferation of glomerular epithelial cells, mainly in Bowman's capsule. Numerous crescents, composed mainly of epithelial cells, and moderate pseudotubule formation were seen (Fig. 4). A large number of glomeruli showed focal fibrinoid necrosis with fibrin thrombi distending one or more capillary loops (Figs. 5, 6). A few glomeruli were normal, and a small number were scarred and hyalinized. All except the least altered glomeruli showed fibrillar deposits of P.A.S.-staining material in the centrilobular areas of the tufts. There was very little endothelial proliferation. Extensive moderate tubular atrophy was seen. Red cell and hemoglobin casts were in moderate numbers.

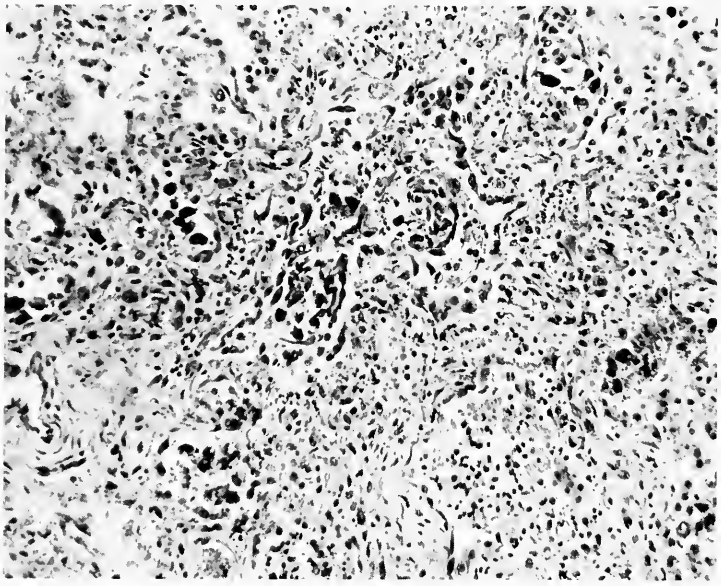

Figure 3. Case 4. Fibrous thickening of alveolar walls. Darkly stained rounded cells in the alveolar spaces contain hemosiderin. They are mixed with iron negative polygonal and flattened cells which appear to be desquamated alveolar lining cells. Iron stain. X 180.

Discussion

The renal lesions in cases 2 and 3 are characteristic of chronic glomerulonephritis, with granular kidneys which were markedly contracted in case 3. The only glomerular changes in cases 2 and 3 which appear noteworthy, because they also appear in the other two cases, were pseudotubule formation in Bowman's capsular space of some unscarred glomeruli. However, the renal changes in cases 2 and 3 are indistinguishable from other causes of chronic glomerulonephritis (Table 2).

The renal changes in cases 1 and 4 are characterized by diffuse, marked proliferation of epithelial cells of Bowman's capsule, with formation of crescents (Figs. 1, 4). This feature was emphasized in three cases of Goodpasture's syndrome reported by Canfield, *et al.*[7] and appears in the description of renal changes in a majority of case reports. The conventional label which could be applied to these

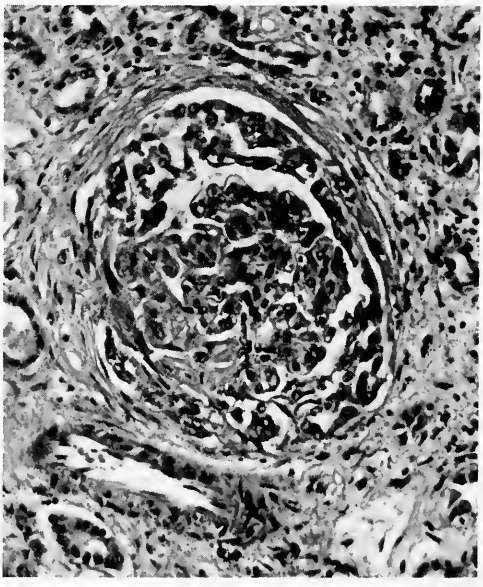

Figure 4. Case 4. Glomerular epithelial and fibrous crescent. The glomerular tuft appears nearly normal, except for adhesion to Bowman's capsule. H and E. X 350.

Table II Comparison of Glomerular Lesions in Goodpasture's Syndrome with Glomerulonephritis and the Nephrotic Syndrome (% of Glomeruli Examined)

	Goodpasture's Syndrome				Nephrotic Syndrome with Nephritis			
	"Subacute"		Chronic		Proliferative		Chronic	
Case	1	4	2	3	Post-mortem (2 patients)	Biopsies (21 patients)	Post-mortem	Biopsies (7 patients)
Glomerular Capillary Thrombi	0	40	0	0	0/0	0	0	0
Endothelial Proliferation	40	0	10	0	65/60	50–100 mean 76	0 0	0–30 mean 16
Crescents	90	60	0	5	5/5	0–80 mean 6	0	0–10 mean 1
Scarred Glomeruli	0	10	75	95	25/0	0–15 mean 4	30	7–71 mean 38

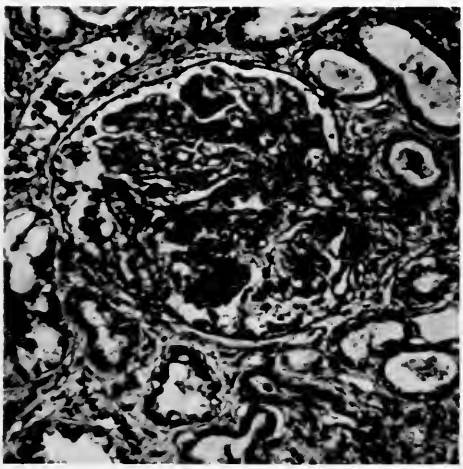

Figure 5. Case 4. Small epithelial crescent. Amorphous deposits in the lumen and walls of glomerular capillaries, interpreted as fibrin thrombi and focal fibrinoid necrosis. H and E. X 267.

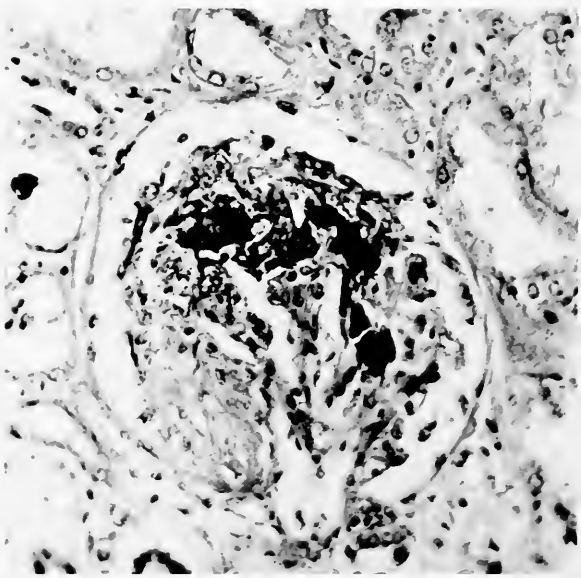

Figure 6. Case 4. Patches of fibrin or fibrinoid necrosis in glomerular tuft, staining intensely with phosphotungstic acid hematoxylin. X 367.

large smooth kidneys is "subacute glomerulonephritis." However, the glomerular tufts, especially in case 4, show little endothelial proliferation (Fig. 4), an observation which has been made previously in some cases of Goodpasture's syndrome.[8] The nephrotic syndrome, associated by many authors of pathology textbooks with "subacute glomerulonephritis," was absent, as it appears to have been in most, although not all, cases of Goodpasture's syndrome.[9] The significance of this observation is emphasized by comparison with the number of glo-

meruli showing various lesions in kidneys of patients with the nephrotic syndrome associated with proliferative and chronic glomerulonephritis, examined at this hospital (Table 2).

This shows no difference in the glomerular changes of chronic glomerulonephritis associated with the two syndromes, except for a somewhat higher proportion of scarred glomeruli in Goodpasture's syndrome. However, a comparison of glomerular changes, both in "subacute glomerulonephritis" associated with Goodpasture's syndrome and in proliferative glomerulonephritis associated with the nephrotic syndrome, shows a lower frequency of glomerular endothelial proliferation in Goodpasture's syndrome and a much higher frequency of crescents.

The pulmonary changes in cases 1 and 2 are not remarkable except for the extent and degree of recent and old alveolar hemorrhage. Cases 3 and 4 show, in addition, fibrous organization of alveolar fribrinous exudates and proliferation of alveolar lining cells. In case 4 the alveolar exudate in many areas resembled that illustrated in desquamative interstitial pneumonia[10] except for the presence of hemosiderin-containing macrophages in addition to iron-free rounded or flattened cells (Fig. 3). Independent examination of the lung and kidney slides in case 4 by two members of the Pathology Department led to the suggestion that very similar changes were occurring in the alveolar lining cells and in the epithelial lining cells of Bowman's capsule. A similar epithelial proliferation was present in the pulmonary alveoli in case 3 and in the glomerular epithelium in case 1. It is possible that changes in the epithelial cells of the glomerular tufts could alter the capillary basement membranes, leading to hematuria. Marking the lamina densa of rats with silver given as silver nitrate in drinking water has implicated the epithelial cell as the source of the glomerular lamina densa.[11]

Four pathogenetic or etiologic possibilities are considered:

1. Goodpasture's syndrome may result from infection by a virus with an affinity for alveolar and glomerular epithelial cells, analogous to the affinity of cytomegalic virus for aveolar lining cells and renal tubular epithelium. Viral hepatitis is an example of an infection of visceral cells of epithelial origin which may lead to progressive chronic inflammatory changes with regeneration. No viral inclusions were seen in the present cases.

2. Goodpasture's syndrome may result from exposure of alveolar and glomerular epithelium to the same toxic substance, analogous to the probable carcinogenic effect of cigarette smoking on the epithelium of both the bronchi and the bladder. Such a toxin might be inhaled, or ingested and excreted by both organs, like alcohol. One substance which has been reported to cause chronic renal lesions characterized by marked proliferation of the glomerular epithelial cells is saccharated iron oxide, in rabbits given repeated intravenous injections.[12] However, this glomerular siderosis was not found in animals given saccharated iron oxide intravenously. Some observers regard Goodpasture's syndrome as a complication of idiopathic pulmonary hemosiderosis.[13] Occupational exposure to gasoline has been reported in some patients with I. P. H.,[14] but no such occupational or any know toxic exposure was found in these four patients.

3. The glomerular lesions may result from capillary thrombosis, such as was seen in case 4 (Fig. 5). A few alveolar capillary thrombi were also seen in this case. However, the glomerular thrombi were generally associated with foci of

fibrinoid necrosis in the adjacent area of tuft (Fig. 6) and are probably secondary to focal glomerular necrosis. There is a good possibility that fibrinoid necrosis in glomeruli is the earliest renal lesion in Goodpasture's syndrome. This lesion may cause both hematuria and proliferation of Bowman's capsular epithelium which persists after the necrotizing process subsides.

4. The pulmonary and glomerular lesions may result from an auto-immune reaction between serum antibodies and a common antigenic component of alveolar and glomerular tissue. Gamma globulin has been demonstrated in the alveolar septa as well as in the basement membranes of glomeruli from one patient with Goodpasture's syndrome.[15] Duncan, *et al.*[16] found that gamma globulin was localized to the glomerular capillary basement membrane from three patients, and beta $_{1c}$ globulin was also present at this site in one of their patients with Goodpasture's syndrome. Lung tissue from this patient showed no deposition of either gamma or beta $_{1c}$ globulin. There is experimental evidence that the basement membrane of glomerular capillaries and alveolar walls are antigenically related.[17] However, pulmonary hemorrhages are not usually produced by injecting anti-lung serum intravenously. Also, gamma globulin has been demonstrated along the glomerular basement membrane by the fluorescent antibody technique in a large number of different human diseases. It seems unlikely that glomerular deposition of antibodies to basement membrane is the cause of all the dissimilar renal lesions that occur in systemic lupus erythematosis, amyloidois, diabetes millitus and several types of glomerulonephritis. Good, *et al.*[18] have recently reported that immune gamma globulin and beta $_{1c}$ globulin are deposited in a linear fashion along the glomerular basement membranes in Goodpasture's syndrome, unlike the lumpy, bumpy deposits seen in other forms of glomerulonephritis. This linear distribution resembles the experimental nephritides caused by anti-glomerular antibodies and differs from the lumpy deposition of antigen, antibody and complement along the outer aspect of the basement membrane in nephritides caused by circulating antigen-antibody complexes.[19]

The most likely pathogenetic sequence appears to be:

1. The alveolar capillary basement membranes are damaged by an unknown infective or toxic agent.

2. In sensitive individuals, serum antibodies form to alevolar basement membrane which cross react with the glomerular basement membrane and fix complement.

3. The earliest renal lesion is glomerular capillary necrosis and thrombosis, followed by proliferation of epithelial cells of Bowman's capsule.

4. Some patients survive long enough to develop a histologically nonspecific chronic glomerulonephritis.

It is unlikely that antibodies to glomerular basement membrane can be demonstrated in the serum of a patient with Goodpasture's syndrome, because such antibodies would be rapidly taken up by the excess antigen of the total glomerular capillary bed. If any patient with this usually fatal disease is treated by renal transplantation, the possibility should be considered that continued antibody formation will damage the grafted kidney, but by a different mechanism than the usual process of graft rejection. Hyperacute rejection of kidney allografts, associated with pre-existing humoral antibodies against donor cells and including complement-fixing kidney antibodies, has been reported in two patients suffering

from chronic pyelonephritis and chronic glomerulonephritis, respectively.[20]

A similar humoral antibody rejection of renal grafts in Goodpasture's syndrome might be avoided by a period of hemodialysis after bilateral nephrectomy and prior to renal grafting. During this period, if the disease were active the presence of antibodies in the serum could be demonstrated by fluorescein conjugation and staining of glomerular basement membranes in sections of normal human kidney or animal kidney, since such antibodies are not specific species. Such antibodies might be expected to increase in quantity for a few days and then, hopefully, to decline gradually to a sufficiently low level to permit grafting.

Summary and Conclusion

The kidneys of two patients with Goodpasture's syndrome showed a nonspecific chronic glomerulonephritis at autopsy. In the other two cases there was glomerulonephritis charatcerized by epithelial crescents, and a lesser degree of glomerular endothelial proliferation than occurred in patients with glomerulonephritis who did not have hemoptysis. In two of the four cases there was alveolar epithelial proliferation and desquamation in addition to the accumulation of hemosiderin-pigmented macrophages in alveoli.

It appears that a distinctive type of glomerulonephritis may occur in Goodpasture's syndrome. Some patients survive long enough to develop a histologically nonspecific chronic glomerulonephritis. If the renal lesions of Goodpasture's syndrome are caused by antibodies to glomerular basement membrane, such antibodies could be demonstrated in the serum after bilateral nephrectomy, and renal transplantation might have to be postponed until they disappeared.

Case 3 is the first known occurrence of Goodpasture's syndrome in a Negro.

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Book Reviews

Development of the Lung (Ciba Foundation Symposium) edited by A. V. S. de Reuck and R. Porter. i-xiii and 1-408 pp. 1967. Little, Brown & Co., Boston, Mass., price $13.00.

The uniformly high quality of the familiar series of Ciba Symposia is apparent again in this new volume. It is an important addition to two products of earlier meetings: "Problems of Pulmonary Circulation" (November, 1960) and "Pulmonary Structure and Function" (July, 1961). Having participated in the second of these symposia, I can attest, personally, to the special value to the participants of the lively, informative discussions which follow each formal presentation and to the readers, of the verbatim reporting of these discussions.

The present volume includes the papers and discussions of some 25 recognized authorities on pulmonary anatomy, physiology, and pathology. They were drawn from the *Who's Who* of pulmonary research and, thus, guarantee the authority and high quality of this compilation of the most recent work and thought on lung development. The participants in the symposium were assembled from Czechoslovakia, Germany, Great Britain, France, Sweden, Switzerland and the U.S.A.

Lung development is considered under five principal headings. "Phylogeny of the Lung" includes papers on gas transport from environment to cell, gas transfer in water-breathing dogs, evolution from water to air, and quantitative aspects of vertebrate gas exchange. "Ontogeny of the Lung" covers lung embryology, its postnatal growth, and a general discussion on the critically important surface-active lining layer of the alveoli. "The Gas Liquid Interface" includes physiological consequences of the apposition of blood and gas in the lung and a comprehensive paper and discussion on the alveolar lining layer. Under "Intrauterine Gas Exchange" are discussed comparative properties of lungs and placenta; a graphical analysis of placental gas exchanges, O_2 supply of the fetus, O_2 transport in fetal blood, and O_2 consumption of placenta and fetal membranes of sheep. The final topic: "The Start of Breathing" contrasts pulmonary circulation in the fetus and newborn and discusses liquid uptake from lungs at the initiation of breathing.

The wide scope of the subject matter of this volume is not really suggested by its title. As the Chairman, Dr. P. Hugh-Jones remarked: "It is always interesting to me that findings in one subject are so directly relevant to problems in another. In this symposium we have the satisfying example of the relevance of the hydrogen and hydroxyl ion ratio, realized from study of the physiological evolution of animals, to the practical problem of cooling patients during cardiac surgery." Those who are concerned with pulmonary physiology, the premature nursery, pediatric anesthesiology, and neonatology in general will find this new book most informative and stimulating. Investigators of pulmonary problems would profit not only from the wealth of information provided, but from comments by the participants on areas in which our knowledge is still incomplete and where further research is urgently required.

VERNON E. KRAHL, PH.D.

Dean's LETTER

Dear Members of the Medical Alumni and Students:

Early in medical education it is difficult to impress the student with the variable face that a given disease may present in different patients. The student is trying to master information and criteria necessary to identify specific diseases. The variable picture of a given disease is confusing and the student searches for simple answers that will make diagnosis easier. While this is a rewarding effort in some instances, it only decreases the problem but does not eliminate it.

At Maryland there has been a consistent effort in the medical education program to give the student considerable contact with patients so that they may learn firsthand about the variable nature of specific diseases. The faculty has felt that this is an important part of medical education in the development of judgment in patient diagnosis and care. Further, it impresses the medical student early in his development that the physician must remain a student throughout his professional career.

In the study and revisions of the curriculum and instruction program, the necessity of a reasonable volume of clinical material for student work-up has been maintained as essential in the education of the physician. The principal aim of the medical schools remains that of preparing the medical student to become an excellent practicing physician.

Sincerely,

WILLIAM S. STONE, M.D.
Dean

Faculty NOTES

Recent Promotions

Dr. Gladys E. Wadsworth, associate professor, department of anatomy; **Dr. Albert F. Heck,** associate profesor, department of neurology; **Dr. Richard S. Mumford** and **Dr. Umberto Villasanta,** associate professors, department of obstetrics and gynecology; **Dr. Richard C. Cavonius** and **Dr. Stanley S. Schocket,** associate professors, department of ophthalmology; **Dr. Leo M. Karpeles,** associate professor, department of physiology; **Dr. George U. Balis, Dr. Jonas R. Rappeport,** and **Dr. Charles Savage,** associate professors, department of psychiatry, The Psychiatry Institute.

Dr. Entwisle Named President of ATPM

The Association of Teachers of Preventive Medicine has elected **Dr. George Entwisle** this year's president. Dr. Entwisle is professor and chairman of the department of preventive medicine and rehabilitation at the University of Maryland School of Medicine.

He joined the faculty of the School of Medicine in 1956 and became department chairman two years later.

Dr. Bloedorn Now Head of Therapeutic Radiology at Tufts

Dr. Fernando G. Bloedorn, formerly Head of the Division of Radiotherapy at the School of Medicine, has been named Professor and Chairman of the Department of Therapeutic Radiology at Tufts Medical Center. Dr. Bloedorn had a long and distinguished career at the School of Medicine being responsible for the development of therapeutic radiology to its present high level. One of his most important contributions was the introduction of the Betatron in the treatment of certain selected malignancies.

Dr. Derbyshire Honored By Student Council

Dr. Robert Derbyshire, associate professor of sociology in psychiatry at the University of Maryland School of Medicine, was honored as faculty member of the year by the Student Council at the School of Medicine.

A member of the faculty since 1961, Dr. Derbyshire teaches introductory psychiatry and is director of undergraduate psychiatric education.

Pathology Professor Establishes Student Fellowship

Dr. Lester Kiefer, pathologist-in-chief of the Memorial Hospital in Cumberland Maryland, visiting Assistant Professor of Pathology in the School of Medicine, and a former full-time faculty member, has established an annual student fellowship in pathology according to information released by Dr. Robert Schultz, Acting Professor of Pathology. The award will be offered for the first time in 1968.

Drs. Nelson and Merlis Named to Advisory Board

Dr. Erland Nelson, Professor and Head of the Department of Neurology at the University of Maryland, and **Dr. Jerome Merlis,** Professor of Neurology and Clinical Neurophysiology, have been named to the professional advisory board of the Epilepsy Foundation of America. The professional Advisory Board will assume financial responsibility for directing the research, professional education, demonstration projects, and other programs conducted by the Epilepsy Foundation.

Dr. Wells Receives Lederle Grant

Dr. Joseph Wells, Assistant Professor of Anatomy at the School of Medicine, is recipient of 1 of the 12 1968 Lederle Laboratories grants awarded to medical school faculty members for their dedication to medical teaching.

DR. STONE TENDERED COMMEMORATIVE DINNER

More than 400 friends and faculty assembled at dinner on May 9, 1968, to honor Dr. William S. Stone, Dean of the School of Medicine, on the occasion of his retirement. Honored guests included Lt. General and Mrs. Leonard D. Heaton, The Surgeon General, Department of the Army; Dr. and Mrs. Wilson H. Elkins, President of the University of Maryland; Dr. and Mrs. William B. Long, member of the Board of Regents of the University of Maryland; Dr. and Mrs. Albin O. Kuhn, Chancellor of Baltimore Schools, University of Maryland; Dr. and Mrs. Thomas B. Turner, Dean, Johns Hopkins School of Medicine; and Dr. and Mrs. John O. Sharrett, President, Medical Alumni Association. Dr. George H. Yeager, Director of the University of Maryland Hospital, served as Toastmaster. The invocation was given by the Reverend Donald C. Kerr, Minister of the Roland Park Presbyterian Church, Baltimore.

Following an informal reception and a substantial dinner served in a very attractive manner, Dr. Yeager called attention to the long and enviable professional record which has marked the career of Dr. Stone. A page from the program calling attention to his many accomplishments follows:

Born 1902. *Graduate, University of Idaho,* 1924—*B.S.;* 1925—*M.S. Graduate, University of Louisville School of Medicine,* 1929—*M.D. D.SC. (Hon.)* 1946.

Internship, William Beaumont General Hospital, 1929-31; *Army Medical School,* 1931-32; *Surgical Service, Fort McPherson Hospital,* 1932-34. *Chief, Parasitic Disease Division, Army Medical School,* 1934-38. *Army Medical Research Board,* 1938-39. *Pathologist, Gorgas Hospital,* 1940-41. *Chief, Laboratories Division and in charge of research program in Preventive Medicine, Surgeon General's Office U.S. Army,* 1941-43. *Consultant, Preventive Medicine and Chief of Research, North African and Mediterranean Theatres,* 1943-45. *Chief, Preventive Medicine Division, Air Surgeon's Office,* 1945-46. *Chairman, Army Medical Research Board,* 1946-50. *Commandant, Army Medical Service Graduate School,* 1950-54.

Director of Medical Education and Research, University of Maryland, 1954. *Dean, University of Maryland School of Medicine,* 1955. *Diplomate, American Board of Pathology, American Board of Preventive Medicine, U.S. Army representative to National Research Council,* 1946-50, *Committee on Medical Research, Department of Defense. The Halstead Society, District of Columbia Academy of Medicine, Consultant to the Surgeon General, U.S. Army. Chairman, Medical Education for National Defense, A.A.M.C. Sternberg Medalist U.S. Army Medical Corps*-1932. *Sigma Xi. Phi Beta Kappa. Alpha Omega Alpha. President's Science Advisory Committee*-1961. *Field of special interest—Medical Education and Health Services during National Emergencies.*

Brief Testimonials

Substituting for General Heaton, Major General Joe E. Blumberg brought a direct message from General Heaton concerning Dr. Stone's distinguished military record, particularly his activities in World War II, Korean War and his accomplishments as the Director of the Walter Reed Army Institute of Research. General Blumberg spoke particularly of Dr. Stone's ability to organize military research teams which attack certain military medical problems in an orderly and purposeful fashion. He praised him for his foresight and many intangible contributions to military medicine.

Dr. Wilson H. Elkins, President, University of Maryland, first spoke of the Dean's personal qualities.

CAMERA VIEWS

---DEAN STONE TESTIMONIAL

Dean and Mrs. Stone greet the incoming guests.

Dr. Wilson H. Elkins, President of the University of Maryland, speaks of Dean Stone's administrative achievements.

Dr. Thomas B. Turner, member of the Class of 1925, University of Maryland School of Medicine and Dean of the Johns Hopkins University School of Medicine, speaks of his academic associations with Dean Stone.

Major General Joe M. Blumberg, M.C., U.S.A., an old friend of Dean S brought a direct message from Lt. General Leonard D. Heaton, M.C., Surgeon General Department of the Army, concerning Dr. Stone's d guished military record, particularly his activities in World War II, K War and his accomplishments as the Director of the Walter Reed Institute of Research.
From left to right, Mrs. William S. Stone, Dean Stone, General Blum and Dr. George H. Yeager who served as toastmaster.

Dr. Theodore E. Woodward presents Dean Stone with a citation from members of the faculty.

Mrs. William S. Stone is presented a memento of the occasion by Dr Carl Weaver, Assistant Dean.

"It has been said on many occasions and by many people that the first qualification for a good doctor is to be a good man—that is, a man of integrity, broad vision, compassion, and moral strength," he said. "If that is true, as it surely is, then a dean of a medical school must have the same qualities in even greater abundance. These are the qualities that I have admired in Dr. Stone during his 15 years with the university."

Reviewing Dr. Stone's contributions to the medical school, Dr. Elkins spoke particularly of his important role in the redevelopment of the Baltimore campus: "Five years from now the physical plant of the School of Medicine and University Hospital will be developed to the extent of providing for the growing needs of medical education. It will be an imposing complex . . . it will bear the mark of his vision, dedication, and hard work."

Dr. Thomas Turner recalled some of the amusing and trying experiences of World War II and of his happy associations with (then) Col. Stone and his help in overcoming a lack of familiarity with army methods. Dr. Turner recalled with pleasure the many cooperative activities between the Johns Hopkins School of Medicine and the University of Maryland made possible through the good offices of Dr. Stone.

Dr. Theodore E. Woodward, Professor and Head of Medicine, represented the faculty in acknowledging with gratitude the many contributions of Dean Stone to the academic development of the medical school. He spoke as a personal friend of Dean Stone, whom he described as "a modest man, a diligent administrator, a tenacious fighter for principles, and a dedicated dean of the School of Medicine."

Dr. Woodward presented Dean Stone with a small testimonal which he read.

William Spencer Stone, M.D., D.Sc.

Know all men by these presents that;

Throughout a productive medical career, William S. Stone dedicated his life to service. United States Army authorities marked him as a top medical scientist and charged him with direction of programs vital to our national security. He was the distinguished Chief of Research and Preventive Medicine of the North African and Mediterranean theaters, 1943-1945; Chairman, Army Medical Research Board, 1946-1950; Commandant, Walter Reed Army Medical Service Graduate School, 1950-54. Pioneer programs unique for their successful control of typhus fever, malaria and other pestilential diseases are attributable in considerable measure to Col. Stone's able planning and implementation under difficult circumstances.

Under Doctor Stone's leadership as Director of Medical Education and Research in 1954, and Dean of the University of Maryland School of Medicine beginning in 1955, research facilities and faculty personnel expanded in many directions, the postgraduate training program broadened, and the curriculum for undergraduate education improved consonant with contemporary developments. His broad experience in diverse medical fields kept the medical center abreast of national and international trends.

Faculty and students know William Stone as a modest man, a diligent administrator, a tenacious fighter for principles, and as a dedicated dean of the School of Medicine.

From faculty and friends of the University of Maryland School of Medicine and Hospital on this ninth day of May, Nineteen Hundred and Sixty-Eight.

An important personage in the Stone team was recognized in the person of Mrs. William S. (Louise) Stone who was greeted by the Toastmaster and, in the name of faculty and friends, presented with a memento of crystal. This was graciously acknowledged.

Prior to the benediction, Dr. Stone expressed with dignity and brevity his appreciation for the tangible expression of respect and appreciation shown him by those in attendance. A solemn benediction closed the event.

Students and Faculty Honor Dean Stone

Mrs. Stone, Dean Stone and Ronald L. Elson

Dr. Theodore E. Woodward

On May 14, 1968, students and senior faculty gathered in the Health Sciences Library auditorium honoring Dean Stone for his accomplishments as a military career officer and as the Head of the School of Medicine. Dr. Theodore E. Woodward spoke in some detail of Dean Stone's accomplishments and particularly of the events relating to his being named Director of Medical Education and Research and Military Dean of the School of Medicine. Dr. Woodward spoke briefly of his military career and of his first meeting (the then Col. Stone in Morocco in 1943) while he was active in research relating to infectious disease control with particular emphasis on mosquito and louse-born diseases. Dr. Woodward then outlined the post-war era when Col. Stone became commanding officer of the Walter Reed Army Institute of Research and of the pressing needs of the School of Medicine of the University of Maryland for forceful leadership in research and teaching. He narrated of when a mutual friend, the late Joseph Smidell, who proposed to the late Judge William P. Cole Dr. Stone's appointment and of how in 1955 he succeeded Dean H. Boyd Wylie following his retirement. Other accomplishments of the Dean have included his civic interests of the Baltimore City Board of Education and of his participation in other civic activities, including the work of the American Cancer Society. An expression of gratitude and appreciation was directed to Mrs. William S. Stone who was applauded by the audience.

Ronald L. Elson, third year medical student and president of the Student Council, presented identical bronze plaques to the medical school and to Dean Stone. The plaques bore the dean's name and the inscription "In appreciation from the physicians and students you have guided."

The medical school's plaque will be placed on the wall in Davidge Hall.

One World—Your World*

MATTHEW TAYBACK, Sc.D.†

That we live in an age of crisis is self-evident. Whether the magnitude and frequency of social turbulence in our time is particularly different from that of the past—I doubt. Yet there is an uneasiness among the people and a growing sense of insecurity that leaves no room for complacency and results in a considerable urgency to wrestle vigorously with the problems of our day. Unquestionably, many if not all of you have a sense of having been subjected to a daily diet of crisis these past four years. You will agree, however, that these are of a parochial nature, good for stiffening one's resolve, and preparation for the more fundamental issues which now loom on the horizon.

Whatever your immediate past experience, and that of this nation, these paths now join as you move on to participate in the mainstream of the life of American society. In this regard, I want to share with you my observations, as a health professional, in respect to three areas of considerable controversy about which you will need to form a judgment. These areas involve (1) the posture of the U.S. *vis à vis* the struggle of nations, comprising two-thirds of the world's population, which seek to improve their social and economic structure, (2) the nation's posture *vis à vis* the struggle of a minority, albeit 30,000,000 persons in number, to achieve a minimum acceptable economic and social position, and (3) the posture of medicine as the public seeks to organize and determine the direction of the health industry.

In these days of dismay relative to the tragedy of Vietnam, there is a growing sentiment for disengagement from international relationships. Such sentiment is contrary to the entire historical development of our country. Consider the matter from a medical point of view. The early development of medicine in our country was highly dependent upon a flow of information and of skilled physicians from England and Scotland. Later, the pattern of medicine was influenced by notable scientific advances in France and Germany, while many of the leading American physicians were to complete their post-graduate training in these countries.

In fact then, the lengthy period prior to 1920 was one within which this nation leaned heavily upon the scientific developments overseas. During this time, pleas for isolation and disengagement would have led to stagnation and deprived the American nation of useful medical knowledge.

Since 1940, there has been no diminution of international relationships. However, these have assumed a rather remarkable character. Our medical institutions have become the training center for many of the medical leaders of the developing countries. Our scientific capability has led to pharmacological discoveries of enormous significance for affecting the lives of the 2 billion persons in developing countries. In a related development this same scientific capability has led to chemical and industrial applications to agriculture with the result that food production here far exceeds the needs of our

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* Presented at the Pre-Commencement Exercise of the School of Medicine, Baltimore, June 7, 1968.

† Deputy Commissioner, Baltimore City Health Department.

MATTHEW TAYBACK, Sc.D.

nation's population and can be used to protect the precarious balance between food and population throughout most of the world and thus help eliminate starvation and death as a factor in modern life. The traffic has not been one-way. While we have served as a center of training for scientists and physicians worldwide, many of these have helped us meet our day to day service requirements. It is worth a moment of reflection to note that 15% of new physicians licensed for practice in the U.S. in recent years have come from foreign countries. And this has occurred at a point when our need for medical manpower has proved to be impossible to solve through our own resources and the need for medical assistance overseas has no limit.

Under these circumstances, what justification can there be to deny the assistance of which we are capable, to nations struggling to advance themselves beyond the subsistence level. It could be argued that since we are in deficit in medical manpower, how can we provide the enormous number of trained practitioners which developing nations require? The answer is obvious. We can't. However, the history of man's success in conquering those forces leading to premature loss of life is one which does not exclusively center about the individual efforts of the medical practitioner. The efforts of the political and social scientist and pragmatist have accomplished miracles in improvement and control of the economic and physical environment and with spectacular results in the number of human lives saved thereby and in the vigor and satisfaction assured to these lives.

Among those nations struggling to improve the lot of their masses, a major issue is the proper nutrition of the people. With the simple health measures already introduced, a substantial reduction in death rate has occurred. This has led to an increase in the rate of population growth and thus to a further aggravation of the problem of providing adequate nutritional levels. It is apparent that no fundamental solution of the most elemental need of these people, sufficient food for proper growth and development of children and for effective performance by adults, can be achieved in the absence of attention to the issue of population growth. It is in connection with solution of this problem that we are able to give substantial assistance at modest cost and with enormous benefits.

Our approach is at once, medical, political, and technological. Pursuing a basic philosophy of extending a helping hand to all nations who seek assistance to improve the health status of their people, we learn quickly that, with few exceptions, such nations are faced by a growth

in population beyond their capability to sustain. The growth in population derives from a traditionally high birth rate and a declining death rate. The number of children born to mothers of these nations is more than twice that born to mothers in this country even though careful studies indicate that mothers throughout the world are alike in the size of family they desire.

Gross evidence of malnourishment is rarely found among the first born and second born child. When it does occur, it is among the later born children, and suggests a breakdown in the economic welfare of a family. A review of this situation with appropriate political leaders and with the point of view of sustaining the morale of the family, avoiding the tragic loss of life of young children due to malnutrition, and preserving the health of mothers inevitably leads to affirmative decision to formulate a population policy. The means of intervention to achieve a planned family must be a decision for each family and nation to make in accordance with individual ethics and national mores. To the extent that our assistance is sought, we have the obligation of spelling out the options as they are known to us medically. The choice of options is rightfully a political issue not within our realm. Once a choice is made, it is a matter well within our technical competence and available manpower to provide the basic materials and knowledge by which rapid change can be accomplished in permitting the developing nations to improve the standard of living of their masses. Such an improvement in standard of living has immediate impact upon the health level of these people.

Thus the world beyond our borders faces a crisis of sheer survival of its masses, a crisis commonly known as "the population explosion." The strategy of intervention is known to us and the technical means of assistance are within our capability. Shall we turn our backs because it is none of our business? Shall we hide behind the specious reasoning, that it is not our part of the world which is in trouble? The answer is obvious. Knowledge has been placed in your hands and mine. It is not American knowledge. It is the accumulation of a timeless and boundary-less fund of reasoning. The object of its application can be no less space-limited. We live in one world, and it is your world and mine in respect to our obligation for action.

As one considers the obligation of a profession to avoid a narrow attitude towards its responsibilities, there is yet the admonition "Charity begins at home." Whatever may be your attitude relative to the strife which accompanies the struggle of the poor to improve themselves one cannot escape the conclusion that an enormous error of omission has been made in this nation's commitment towards meeting the health and medical needs of its people—that is, all of its people.

In considering the physician requirements of our nation, constantly there has been reference to the criterion of what the nation was capable of supporting economically under a fee for service principle. The end result of this form of reasoning is a startling scarcity of qualified physicians available to meet the more fundamental health needs of millions of Americans who were not part of the world of users of medical service in those earlier days of the planning of medical education facilities.

As the poor have become polarized geographically, particularly in the urban metropolises, their deprived status in respect to health and medical services has become painfully apparent. Just a few days ago, I had the experience of walking through a neighborhood of row-type low-rental homes consisting of some

10,000 individuals, in which not one physician was available as a family medical counselor and practitioner. The question of "Who is your family physician?" brought forth the derisive response of "Are you kidding?" There is a sense of abandonment, in respect to medical services, now widely present among millions of disadvantaged Americans. And we would do well to avoid a practice of finding an appropriate excuse for this circumstance. Rather, let us face the fact that too long, the arena within which we have been concerned, the interior of the hospital, the surgical service, the laboratory, and the doctor's office has failed to reach effectively a part of the world in our own country which epidemiologically has the greatest need for health services.

The diagnosis is rather clear. We have a community of professionals. They have organized themselves in a pattern which, in their opinion, permits them maximum efficiency. The theory is that what is optimum for the profession serves the population best. Empirically, the theory has proved grossly deficient insofar as adequate service to the poor, although it has performed creditably in respect to the care of middle income and upper income groups. The alternative means of organization of medical services would be to consider the nature of the population socially, economically and geographically, and to meet the nation's medical service needs accordingly. It is not for us, here, to detail the remedy of this situation. It is, however, an issue which will not rest calmly nor one which can be dismissed as extraneous to your concern.

The past history of conservatism by leading medical spokesmen in the matter of advocacy of essential health needs, which has derived from an attempt to conserve the skills and capabilities of medicine for essential purposes, has been misread by many to indicate an unreasonably narrow attitude, by a profession, to its responsibilities. Our hope is that new generations of physicians, by their concern with the problem of the disadvantaged and with related issues, will change an unfortunate stereotype which persists today.

We have considered two problem areas of the world within which you will soon be called to play your part. We come now to still another intriguing area of conflict and yet one of opportunity provided there is some comprehension of the issues involved. Of the total expenditures for health care which now approximate 35 billions of dollars annually, an increasing proportion is managed by a third party, whether this be a government office; Blue Cross-Blue Shield; or a private insurer. However burdensome the current rate of expenditure of health services, it is not sufficiently adequate to cover the costs of services for such problem areas as dental care; skilled nursing home care and related elements of care; drugs for the aged; the care of the mentally ill, the alcoholic and the narcotic addict; the care of the mentally retarded, the management and rehabilitation of the stroke case, the provision of dialysis for the impaired renal case, etc. Furthermore, there needs to be considered also the desirability of such environmental improvements as atmospheric pollution control, stream pollution control, adequate housing for the aged and poor, engineering of roads and automobiles to reduce the loss of life from automobile accidents, etc.

The strategy for successful intervention involves at least social, economic, political, and medical reasoning as well as actions.

Who constitutes the appropriate authority to consider the many possibilities for increasing the health level of the nation and for indicating the appropriate

direction which public policy should take? Here again, you will be faced by a relationship which heretofore you have not experienced. The scientific, professional area within which you have received your training is but one of several bodies which constitute the structure for making decisions concerning the further development of the health service pattern for the American nation.

We have attained a level of health beyond which further development must rest upon the initiative of the individual who will benefit. Various paths are possible. The duty of the professional is to spell out the options. It is essentially for the consumer representative to make the choice. Through this procedure, the burden of advocacy is shared by the profession with the lay world, a partnership which is consistent with the spirit of our day. Lest my thesis be confused—our difficulty does not derive from a lack of knowledge, but quite the contrary. Enormous possibilities are available to meet age-old and newly created health hazards. Many of these techniques require public decisions for implementation because of their nature and their cost.

By way of conclusion, I would hope that you will recognize the challenge of your time, namely, that the world beckons for your knowledge and consideration. You have benefited by the gregariousness of earlier scientists and practitioners who have not been limited by national boundaries. In the face of overwhelming problems, seek the critical factor which may set forth a series of events leading to fundamental solution. Avoid the constraint of your office or laboratory. Remember that only a selected microcosm of the world comes to you. It is for you to seek actively within the broad community to fully appreciate the need for your services. Finally, the achievement of health as an optimum functional state is not the sole responsibility of the physician. It involves economic and social considerations and political decisions. Learn to share your knowledge graciously with other members of society, so that they may wisely choose among alternative paths to the improvement of a community's health.

Pre-Commencement Exercises

The Pre-Commencement Exercises or Dean's Recognition Day at which time formal hooding ceremonies for the Class of 1968 were carried out were held Friday afternoon, June 7th. Dr. Albin O. Kuhn, Counsellor of Baltimore Campuses, extended official greetings and congratulations to the graduating class.

The ceremonies were held on the lawn of the Psychiatric Institute and were preceded by an academic procession. An address by Dr. Matthew Tayback, Deputy Commissioner, Baltimore City Health Department, entitled "One World—Your World" was the principal Pre-Commencement oratory.

Dean William S. Stone, School of Medicine, then administered the Hippocratic Oath to members of the Class of 1968.

The following honors, certificates and awards were next presented: *Faculty Gold Medal:* John de Courtenay Gelin, Wilmington, Delaware.

Balder Prize for highest degree of academic achievement (Summa Cum Laude): Bruce Lawrence Miller, Baltimore.

Certificates of Honor (Magna Cum Laude): John de Courtenay Gelin, Barry Sheldon Handwerger, Baltimore, Michael John Shack, Baltimore, Charles Ralph Beamon, Norfolk, Virginia.

Certificates of Honor (Cum Laude): Richard Spencer Buddington, Hyattsville, Md., Joel Mayer Cherry, Baltimore, Michael John Deegan, Pennsauken, New Jersey, Douglas Boone Hess, Shady Grove, Pa., Carroll Davis Mahoney, Cheverly, Md., Stephen Allen Stuppler, Silver Spring, Md., Irving Darryl Wolfe, Baltimore.

Dr. Leonard M. Hummel Prize for excellence in internal medicine: Edward John Young, Laurel, Md.

Dr. Harry M. Robinson, Sr., Prize for excellence in dermatology: John de Courtenay Gelin.

Dr. Wayne W. Babcock Prize for excellence in surgery: Franklin Richard Stuart, Baltimore.

Dr. Milton S. Sacks Memorial Award for excellence in medicine and hematology: Samuel Bertram Allison, Newark, Delaware.

Dr. J. Edmund Bradley Pediatric Award: Charles Ralph Beamon.

Dr. Jacob E. Finesinger Prize for excellence in psychiatry: John de Courtenay Gelin.

Following the formalities, tea was served in the courtyard.

PRE-COMMENCEMENT EXERCISES

he sacred oath of Hippocrates, the Class of 1968 joins the long line and becomes the 161st class to graduate from the School of Medicine.

Honors are distributed by Dean Stone.

latthew Tayback, Deputy Health nissioner of Baltimore, delivers 968 Pre-Commencement Address.

Formal investiture with the hood of Doctor of Medicine in the University of Maryland.

Class of 1968 prepares for the academic procession before the portico of Davidge Hall.

Class of 1968 assembles at Davidge Hall for Pre-Commencement Exercises.

Medical Education at Maryland* (1954-1968)

WILLIAM S. STONE, M.D.†

An understanding of a program of medical education can best be obtained if there is an appreciation of the base upon which it has been built.

The medical education program at Maryland is intimately tied up with the support and general public appreciation of its parent university.

In Maryland public awareness of the need for excellence in its State university is a relatively recent development. In fact it could be said that this has been true of the eastern seaboard of the United States. In the development of higher education in the United States the pattern has been emphasis on support of private institutions in the east with less attention to public institutions. The reverse has been true of public universites in the central States and west where there has been strong tax support for State universities with less attention to private education.

In the colonial period the development of education in the United States was patterned after the English and Scottish systems, with Oxford, Cambridge and Edinburgh Universities being the models for higher education. The central core of the curriculum at English universities was classical languages and literatures. In addition, such subjects as Aramaic, Syriac, Hebrew, ethics, politics, physics, mathematics, botany and divinity were studied. The Harvard curriculum of 1723 was much the same, except that more Latin was stressed in the freshman year, metaphysics had been added and botany dropped. The other colonial colleges followed substantially the same kind of curriculum, stressing, as did Harvard, the traditional language arts and philosophy.

This program was the only one leading to a bachelor's degree and was rigidly prescribed for all. There was no concept that the varying interests or professional plans of the individual student should be taken into account in constructing a curriculum. It was felt that there was a fixed and known body of knowledge—the "liberal arts" as they had come down from antiquity via the Middle Ages, Renaissance, and Reformation. This constituted absolute and immutable truth, and it was important that it be absorbed—not criticized or questioned—by every student.

About the only way in which the studies in the early colonial colleges differed from those in contemporary Oxford or Cambridge was in the greater prominence accorded in the New World to the learning of Hebrew. Many colonial scholars regarded that language as being of divine origin and one which would be spoken by the saints in heaven. President Ezra Stiles at Yale, for example, sought earnestly to make all freshmen study Hebrew. When, in 1790, this requirement was abandoned, he tried to reconcile himself to giving Hebrew instruction only to volunteers. Stiles sought to increase interest in the course by informing the students that one of the Psalms he taught them would be the first they should hear sung in heaven; he would be ashamed that any of his pupils on that occasion should be ignorant of the holy language.

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* Read at the June 22, 1968, meeting of the U. of M. Chapter Alpha Omega Alpha Honorary Medical Fraternity.

† Dean, School of Medicine, University of Maryland, Baltimore.

Initially the attainment of a college preparatory education was based on apprenticeship in which the student went in residence usually with a minister, a doctor or a lawyer, and for a fee or indentured services was tutored until the instructor decided he was ready to attend college. The college entrance examination was primarily a reading and comprehension test in Latin and Greek. In New England Latin grammar schools were established at an early date and were the equivalent of our primary and secondary schools. Arithmetic was not a college entrance requirement until 1745. By 1870 colleges such as Yale required a knowledge of mathematics, geography, history, and English, in addition to Latin and Greek for admission.

College educational programs were of the classical type with emphasis on Latin, Greek, and philosophy.

In 1715 Yale introduced Newtonian Science and by 1750 almost all colleges taught natural science. Thomas Jefferson first advocated in 1776 that the classical curriculum be supplemented with modern subjects such as history, government, science and modern language.

Considering present day standards, most colonial college educational programs would be classified to be at the secondary school level and not higher education. Their aim was primarily education for the ministry. During the period 1700-1750 50% of college graduates entered the ministry; by 1900 only 6.5% followed this career. Literate college trained clergy were probably the most important single factor explaining the founding of colonial colleges.

The university which Thomas Jefferson established at Charlottesville in Virginia was America's first real State university. It is an authentic example of this type for a number of reasons. First of all, it aimed from the beginning to give more advanced instruction than the existing colleges, to permit students to specialize and to enjoy the privileges of course and subject election. Its course of study when it opened for instruction in 1825 was much broader than that which was customary at the time. Secondly the University of Virginia was by the express intent of its constitution a thoroughly public enterprise, rather than a private or quasi-public one. Finally, its early orientation was distinctly and purposely secular and nondenominational. In all of this, it represented the most thoroughgoing embodiment of the "revolutionary" spirit of the Enlightenment to be found in American higher education during the first decades of the 19th century.

From the beginning, the institution was placed in the hands of a Board of Visitors, directly appointed by the governor and confirmed by the legislature. In the hands of these Visitors were lodged all powers which had been customarily exercised by incorporated boards of trustees. In addition, the State of Virginia made a large investment in the original buildings, library, and equipment of the university, and continued regularly to give it an annual appropriation of money to support its work.

Although the ideas of Jefferson were responsible for the creation of the first State university in the United States, other States in the East and South did not accept this leadership but proceeded to develop their State colleges and universities on the private or quasi-public pattern and many were public institutions of higher education in name only, such as the University of Pennsylvania and the University of Maryland. Following the Civil War, Jefferson's concept of the State university received great attention in the central and western States. When U. S. legislation was passed under the Morrill and the Hatch acts, making it possible,

through land grants, to give greater support to State colleges and universities, our current concepts of the land grant colleges were born in the central and western States. These land grant colleges were among the first institutions of learning in the United States to welcome applied science and the mechanic arts and to give these subjects a recognized place in the college curriculum. They fostered the emancipation of American higher education from a purely classical and formalistic tradition. President Welch of the Iowa State Agricultural College expressed this pragmatic philosophy in 1871 when he asserted "that knowledge should be taught for its uses; that culture is an incidental result." The purpose of Iowa State was defined as being that of promoting "the liberal and practical education of the industrial classes in the several pursuits and professions of life."

Finally, these colleges stood preeminently for the principle, increasingly so important in the middle of the 20th century, that every American citizen is entitled to receive some form of higher education. With the first State universities and municipal colleges, the early land grant colleges represented the force of democracy working as a mighty leaven in the world of American higher learning.

The University of Maryland, although chartered as a State university, operated as a private institution with little State support until the period following World War II.

Its history reveals that on December 18, 1807, the Maryland Legislature authorized the establishment of a medical school in Baltimore. In 1812 the medical college in Baltimore was given legislative authority to add colleges of liberal arts, law, and divinity, and to assume the name, University of Maryland. The University Charter was enlightened, nonsectarian, and democratic in its concepts. However, there was no indication that tax sources could be used in financing the University. Tuition was considered as the principal source of support for the educational programs.

This lack of tax support persisted for the medical school even though the University was reorganized in 1920 by combining the Baltimore schools with the Maryland Agricultural College at College Park. As late as 1942 the State appropriated only $53,559.00 for the operation of the medical school; $34,286.00 of this amount was transferred to the University for overhead support of the medical school, leaving only $19,273.00 available from State appropriation for operation of the Medical School. Tuition was almost the total source of funds available to support the medical education program.

In 1954 the State appropriation for the medical school had only risen to $367,681.00 of which $41,441.83 was allocated to University overhead. This support was less than 1/5 the amount provided for stated medical schools, such as those at Michigan, Iowa, or Illinois, whose State universities have received, throughout most of their existence, strong tax support for their physical plant and educational programs.

This failure of the State to support the medical education program at Maryland, we believe, can be attributed to the long standing concept in Maryland and States on the east coast that higher education should be supported from private sources and not from a tax base. There was also a reluctance on the part of the medical school to ask for State support for its operating budget because of the fear of political interference in its educational programs.

Since 1954 a consistent effort has been made to gain State support for medical education at Maryland. This has been rewarding and during 1965-66 the State

appropriated for the operation of the medical school $1,644,844.00 and in addition $872,437.00 for University overhead for support of the medical school. This is over a four-fold increase in State appropriation in the last ten years.

There are certain basic concepts a medical school must adopt in planning its programs of education. They are:

1. To provide for its undergraduate students the opportunity to acquire a sound, basic education in medicine and foster the development of life-long habits of scholarship. The medical graduate must be a knower but he also must be a doer. He must be scholar in maintaining and developing his knowledge of medicine but he must also constantly develop his skills and judgment and sharpen their application in the delivery of medical care.

2. To contribute to the advancement of knowledge through research.

3. To contribute to the development of teachers, investigators and practitioners through programs of graduate education including residency training.

4. To provide leadership in the programs of continuing education for practicing physicians.

5. To participate in the educational programs of other professions and technical personnel in the health fields.

These basic concepts have been adopted at Maryland and were approved by the President and Board of Regents in the Medical School's Faculty Organization in October 1956.

There are two primary problems to which medical school faculties must give major consideration in their programs of medical education:

1. All medical graduates must be competent to diagnose and treat the sick and injured and provide counsel and assistance in the prevention of these conditions.

2. The medical education program must provide a sound foundation for the physician's future development.

The first problem is created by the fact that there is no reasonably accurate way at present for patients to classify their condition prior to seeking medical care. The first physician contacted and those that may see the patient subsequently must carefully diagnose the condition present before proceeding with treatment. This requires a broad medical background and the development of considerable judgment in solving the patient's problem. Because symptoms are usually not due to a single cause, disease and injury states may simulate one another as well as be present as a complication. For this reason the medical student should have a core of factual knowledge and experience that will allow him to appreciate the disease factors that may be present and the differential ability to eliminate those conditions that are not present. For example, precardial pain may be due to cardiac ischemia, hiatus hernia, gastritis, colitis, gastrointestinal distension, pleurisy, pneumonia, cancer, and a number of other conditions. It is obvious the treatment of these conditions varies and the correct diagnosis must be reached if the patient is to be helped by the treatment. The ability to diagnose is not only required of the primary physician but also of the specialist to whom the patient is referred. The diagnosis that led to the referral may be in error and the specialist must be certain that the patient is properly treated.

The necessity to properly diagnose the patient's condition dictates that core information taught in the four years of medical school be broadbased, up to date in accuracy and the foundation of medical knowledge of every medical graduate. Further, to appreciate the varying picture of a given disease in different patients,

the medical student should have a fairly large patient contact during the clinical years of his medical education.

If the core curriculum is wisely chosen by the faculty and effectively presented, the second problem, namely the sound base for future development, is also accomplished. These facts explain why the core curricula of the four years of medicine are designed as the common base for the development of all fields of medical practice. The development of clinical medical practice knowledge and skills for general practice and the other medical specialties occur in the intern and residency years.

By providing some free and elective time in the curricula, the faculty make it possible for the individual student to explore areas of medicine of particular interest to the student to help him in choosing his career pattern.

These are the guiding principles for the medical education program at Maryland. Activities to accomplish these objectives during the past 14 years have been focused on—

1. Development of an outstanding full-time faculty.

2. Adoption of a core curricula, what should be taught, how should it be taught, what methods should be used to measure students' progress and improve faculty teaching. In addition to the core curricula how much free and elective time can be profitably allowed for the student to develop his own interests.

3. How can we improve the environment for the educational programs by additions and remodeling of the physical plant.

4. How can we obtain sufficient University and State backing to provide the needed resources.

In medicine there are roughly two types of medical schools:

1. Schools that teach by core subject presentation and a minimum of direct patient contact, with observation of patients being demonstrated by faculty. This is exemplified by schools such as the University of Pennsylvania at Hershey, and

2. Schools that teach with basic core subject presentation and substantial student direct patient contact under faculty supervision. Maryland is the latter type school. We consider primary contact with a variety of patients as one of the essentials of an excellent medical education program.

Since 1953 faculty studies and outside consultants hired to assess medical education needs at Maryland have recognized that major improvements must be made.

As a result of these actions the following steps were taken in reorganizing and developing the medical education program.

1. An urban renewal project was requested to be backed by the University and its Regents to give space for development of the medical school and University Hospital.

2. Faculty planning committees were used to determine the physical plant needs and curriculum and instruction changes desired.

3. Budgets were sought to support an adequate full-time faculty in both clinical and basic science years.

4. The medical school's by-laws and those of University Hospital were revised by faculty committees and approved by the University's president and the Board of Regents.

5. Written agreements were worked out with the medical school's affiliated hospitals.

6. A medical service plan was worked out with the University for basic policies on the employment of medical school faculty.

7. The University Hospital Director was made responsible to the Dean of the

Medical School for the operation of the hospital in support of the programs of medical education and patient care.

8. A full-time faculty was recruited for the clinical departments.

9. A faculty curriculum and instruction committee was appointed to work on improvement of the medical education program.

10. Support was given for the construction of a new Health Sciences Library.

11. Remodeling of University Hospital's X-ray, Ob. & Gyn. departments, central sterile supply, operating rooms, elevators and kitchen was instituted. To provide better balance in clinical teaching resources temporary use of some psychiatric beds was diverted to surgery and the 12th floor of University Hospital converted into patient beds.

12. A division of radiation therapy in the Department of Radiology was created and support for cobalt 60 and betatron equipment obtained.

13. A basic science building requested which resulted in the obtaining of Howard Hall and its remodeling in the Basic Science departments with multidisciplined laboratories for student teaching.

14. Plans were made for a North Hospital Building to replace the Outpatient Department and add new beds and other clinical teaching resources. This building was financed through legislative action and federal grants, and will provide for new clinics, 250 new teaching beds, and better services and facilities for medical students, house officers, and para-medical personnel.

15. Support was given for the development of a Student Union Building.

15a. Negotiations were conducted to have the State Medical Examiner's laboratories built on the Baltimore campus to add better resources for the teaching of pathology and aid in clinical research.

16. A program of surgical teaching and research in the shock trauma field was initiated and supported.

17. Studies were carried out with the aid of the A.A.M.C. to determine the medical school's resources in faculty, faculty attitudes, student quality, physical plant, budgetary support, research and administrative leadership. The summary of this study was brought together at a summer faculty meeting with A.A.M.C. representatives at Airlie House, Virginia, in 1963. This led to a planned continuing study of curriculum and instruction with revisions made and reviewed on a yearly basis. In essence the curriculum was revised with less departmental boundaries, more student elective time and instruction on a team basis using faculty from all departments as it appeared most helpful. There was much greater coordination of instruction and less unplanned duplication.

18. A study of Maryland's needs for medical education and research was initiated with the State Planning Commission. The study was completed and published in 1962. Its recommendations were accepted by the University of Maryland through activities of the Hornbake Committee and actions to implement these recommendations were undertaken.

19. Plans were made for an addition to Howard Hall to allow expansion of the basic science faculty to teach classes of 155 medical students by 1970 and to provide research space for the clinical faculty.

20. Requests were made for construction of a high-rise apartment building for graduate students, house officers and junior faculty. Authorization for a three million dollar bond issue has been received from the State for this project and it is now in the planning stage.

21. Negotiations were carried out with Johns Hopkins University Medical School

and the United States Veterans Bureau to construct a 500-bed general hospital adjacent to the University of Maryland campus. These actions were approved by Johns Hopkins and the U. S. Veterans Bureau with construction expected to be started in 1971.

22. Negotiations were conducted with the State Department of Mental Hygiene to construct a mental health center adjacent to the Baltimore campus, between Fayette and Lexington Streets, to be used in the medical student house officer and health ancillary professional education programs.

23. Plans were made and actions taken to implement a Maryland Regional Program in heart disease, cancer, and stroke in conjunction with the State Health Department and Johns Hopkins Medical School.

24. Plans have been supported for additional parking for the Baltimore Campus with the goal of parking facilities for 2500 cars.

25. The medical school has actively worked to obtain resources for support of a para-medical education program in Physical Therapy, Medical Laboratory Technology, X-ray Technology, Occupational Therapy, and Clinical Technology. It is expected that this program will be fully instituted when the North Hospital Building is completed. It will be housed in the old Pathology Building and in space freed by the Dental School when it moves to new quarters.

26. In 1954 there were approximately $300,000.00 research project grants active in the School of Medicine. Only six departments were actively involved in basic research activities. In 1966-67 there was over $8,000,000.00 in active research grants with all 17 departments and the Institute of International Medicine involved in basic and clinical research. This is a 27-fold increase in 12 years.

In conclusion, I believe that at present both the University and the State are more cognizant of the needs for medical education in Maryland and steps are being taken to develop this Medical School into a major medical education center.

WILLIAM S. STONE, M.D.

May 22, 1968

Bubacker, John S. and Rudy, Willis, *Higher Education in Transition,* First Edition, Harper and Brothers, New York, 1958.

ALUMNI ASSOCIATION SECTION

President's Letter

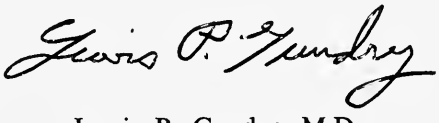
Dear Fellow Alumni:

A new year is starting for your Board of Directors and President. First, let me say that John Sharrett, your outgoing president, with his dedicated, energetic, hard-working approach, presents a very difficult "act" to follow. Jack deserves a vote of thanks from all of us for a job well done. To use a trite expression, we will do our best.

The most important and interesting problem which has been passed along to the present administration is the idea of restoring Davidge Hall. This wonderful old edifice is the oldest medical school building continuously in use in the teaching of medicine in the country.

It was the opinion of the Board of Directors that Davidge Hall should be established as a National Shrine. This would be a joint project with the University of Maryland Medical School.

Many Alumni have expressed interest in this undertaking and any suggestions will be welcome. Incidentally, as this is written, a picture of Davidge Hall appears on the cover of the September *Maryland State Medical Journal.*



Lewis P. Gundry, M.D.
President

ALUMNI JUNE WEEK 1968

Medical Alumni Association—Medical, Surgical, and Pediatric Association Meetings a Success

June week 1968, was an important one on the University of Maryland campus. Since this year, it comprised not only the annual meeting of the Medical Alumni Association but also the University of Maryland Surgical Society, the University of Maryland Hospital Medical Association, the Douglass Obstetrical and Gynecological Society and the Bradley Pediatric Society.

Beginning on June 5th, with a reception at the Lord Baltimore Hotel and numerous class reunions, registration was held in the morning of June 6th, at Davidge Hall. Annual meetings of the several scientific societies followed.

Annual Alumni Business Meeting

The annual business meeting of the Medical Alumni Association in Chemical Hall was called to order at 12:10 by the President, Dr. John O. Sharrett. He briefly summarized the activities and endeavors of the Board of Directors during the year May 1, 1967 to April 30, 1968.

Dean William S. Stone, Dean of the School of Medicine, welcomed the members of the Medical Alumni Association. He asked them to be cognizant of the fact that the coming year is not going to be easy financially for the School of Medicine. The School will need the support of every alumnus during this year of austerity.

The treasurer, Dr. Robert B. Goldstein, reported a balance in hand in the amount of $51,505.88.

The Necrology was read by Dr. William H. Triplett, Executive Director. A moment of silence and respect was observed for the following Alumni who had died during the year 1967-68.

NECROLOGY

Class of 1897

Love, Robert W.
MacDonald, Claude D. J. (P&S)

Class of 1902

Singewald, Albert G.

Class of 1903

Evans, John (BMC)

Class of 1904

Mock, David C. (P&S)
Cohen, Morris D. (P&S)

Class of 1905

Jenkins, Harry E.
Magarian, S. Malcolm (BMC)
Kasson, Benjamin B. (P&S)

Class of 1906

Playse, Linn F. (BMC)
Roemer, Jacob (BMC)
Healy, Edward F. (BMC)
Carlton, Romulus L.

Class of 1907

Carr, Walter A. (P&S)
Mikkelsen, Peter C. (BMC)
Perry, Ernest M. (P&S)

Class of 1908

Pratt, Ivan E. (P&S)
Conn, Clyde W. (P&S)
DePasquale, James (BMC)
Stevenson, Thomas W. (P&S)
Matheke, Otto G. Sr. (P&S)

Class of 1909

Priest, Wilmer M.
Phillips, William G. (P&S)
Hill, William G. C. (P&S)

Class of 1910

Grisinger, George F. (P&S)
Weible, Earl B. (BMC)
Rubin, Louis
Foster, Herbert M.
Daly, Charles W. (P&S)

Class of 1911

Hogan, John F. (P&S)
Wilkins, Java C.

Class of 1912

Whitaker, Edwin V.
Greenstein, Charles J. (BMC)
Lenzner, Simon G.
Mendeloff, Morris I. (P&S)

Class of 1913
FINNERTY, CHARLES W. (P&S)
MARTIN, WILLIAM T.
DIXON, JAMES S. (P&S)

Class of 1915
KERKOW, ROY R.
GONZALEZ, LUIS F. (P&S)
JENKINS, WILLIAM H.
MILLER, WILLIAM C.

Class of 1916
BRAY, THOMAS L.
VAN POOLE, CARL M.

Class of 1917
DAVIDSON, WILLIAM B.

Class of 1918
CARLIN, EDWARD J. M.

Class of 1920
SMITH, FRED B.
HOLDEN, FREDERICK A.
LUEDERS, WILLIAM JR.

Class of 1921
SHUBERT, FELIX S.
SABIN, FREDERICK C.
SHERMAN, SOLOMON

Class of 1922
TRYNIN, AARON H.
WILSON, THOMAS N.
MCCOY, C. GLEN
MONNINGER, ARTHUR C.

Class of 1923
HADDOCK, DOUGLAS A.

Class of 1924
KAFKA, MAXIMILIAN M.

Class of 1926
FREUDER, ARTHUR N.

Class of 1927
KARNS, CLYDE F.
MORRIS, FRANK K.

Class of 1928
GOLDBERG, VICTOR
LERNER, MORRIS
KOTCH, NATHAN H.

Class of 1929
BONGIORNO, HENRY D.
VOLENICK, LEE J.
MURPHY, JOHN EDWARD

Class of 1931
BARTON, PAUL C.
BAUMGARTNER, EUGENE I.
WHIMS, HAROLD C.
JONES, ARTHUR F.
MOYERS, WALDO B.

Class of 1932
MCGOVERN, WILLIAM J.

Class of 1933
HEDGPETH, LOUTHEN R.
WOLBERT, FRANK O.

Class of 1934
SCHWARTZ, THEODORE A.

Class of 1935
KAMINSKY, AARON L.

Class of 1936
DROZD, JOSEPH

Class of 1937
SCARBOROUGH, C. PARKE
GORDON, WILLIAM C.
WOLFF, ELDRIDGE H.

Class of 1940
DWYER, JAMES R.

Class of 1943
EPPERSON, JOHN W.

Class of 1944
FUTTERMAN, PERRY

Class of 1947
MEYER, JANE K.

Class of 1953
DOERNER, WYLAND F., JR.
GARLOCK, FREDERICK A.

Class of 1956
LOVE, THOMAS A.

Thomas B. Turner Class of 1925 Receives Gold Key

The President of the Medical Alumni Association, John O. Sharrett, then recognized Dr. Thomas B. Turner, a member of the Class of 1925, and Dean of the Johns Hopkins University School of Medicine, cited by the Alumni Association as the 1968 recipient of the Honor Award and Gold Key. In presenting this important honor, Dr. Sharrett said (in part).

"Each year we gather at this time to honor one of our alumni who has made outstanding contributions to our profession. It is my pleasure to introduce Dr. Thomas B. Turner, Class of 1925, as recipient of the Honor Award and Gold Key for 1968.

"Dr. Thomas B. Turner was born in Prince Frederick, Maryland. He graduated St. John's College, Annapolis, in 1921 and from the University of Maryland School of Medicine, 1925. His internship residencies were at the Hospital for the Women of Maryland and Mercy Hospital in Baltimore. Briefly, it can be related that Dr. Turner progressed from Instructor in Medicine at the Johns Hopkins Medical School 1928, to Professor of Microbiology, Johns Hopkins University in 1939, and to Dean of the Medical Faculty in 1957, a position he now holds. He served in the U.S. Army from 1942 to 1946, and as a Colonel received the Legion of Merit. His active part in medical societies are numerous and his publications number over 80.

"At this point, I want to digress a minute. I heard Dr. Turner recently comment about his military career and in his unassuming manner, reported that his greatest accomplishment was staying out of trouble and out of the C.O.'s hair.

"As you can imagine, it was with humility that I undertook this introduction and presentation because of my lack of personal knowledge of Dr. Turner and therefore, in the past few months, I have casually questioned his colleagues at Johns Hopkins about him. I feel that we can generalize by reporting that he is portrayed by those who know him socially and professionally as a gentleman, a capable physician, an adept administrator, and a counselor with a patient ear, and ability to bring tranquility to trying situations, having conquered his adversaries and yet leaving them with a sense of victory also.
of victory also.

"I am proud, Dr. Thomas B. Turner, to present you with the University of Maryland Alumni Association Honor Award and Gold Key."

Following the presentation and acclamation, Dr. Turner replied briefly expressing his appreciation for the honor which he accepted with great dignity.

The annual business meeting was then called to order, Dr. Sharrett speaking briefly as follows:

Will the annual Medical Alumni Association please come to order.

First, I would like to say that through the President's Letters in the BULLETIN we have tried to keep you informed of the activities of this society and in review they may quickly be summarized as follows:

1. To review the needs and policy of the BULLETIN in cooperation with the medical school administration and to furnish fiscal support needed.

2. To acquaint some 400 plus members of the Baltimore campus medical community with their opportunity to be active members of this association and their interest has been encouraging.

3. To contact previous recipients of Alumni Student Loans so that repayment would give others an opportunity to benefit from these funds.

4. To initiate a campaign for the permanent preservation of Davidge Hall in its most original known condition as the oldest American medical school building in continual use. From President Elkins, the Board of Regents, and the Dean we have received support, but progress has rightly been curtailed until the next Dean of the Medical School has been appointed.

I would like to take this opportunity to thank all the members for their support this year.

Dr. Sharrett then introduced Dean William S. Stone who gave his annual report on the status of the School of Medicine. This was followed by the report of the Nominating Committee and the election of officers whose names appear below:

President-elect
Dr. Wilfred H. Townshend, Jr. '40

Vice Presidents
Dr. John C. Hamrick, '35
Dr. Herbert Berger '32
Dr. C. Martin Rhode '40

Secretary
Dr. Theodore Kardash '42

Treasurer
Dr. Robert B. Goldstein '54

Executive Director
Dr. William H. Triplett '11BMC

Members of Board 5/58-4/71
Dr. Martin E. Strobel '43
Dr. Henry H. Startzman '50
Dr. Kyle Y. Swisher '48

A nominating committee was duly elected. **Dr. Louis P. Gundry,** the incoming Presi-

dent for 1969, was introduced. The meeting was then adjourned, Alumni members being entertained at a luncheon in the gymnasium, 5th floor, Psychiatric Institute.

Social events occupied the afternoon with many of the class reunions holding special events of their own.

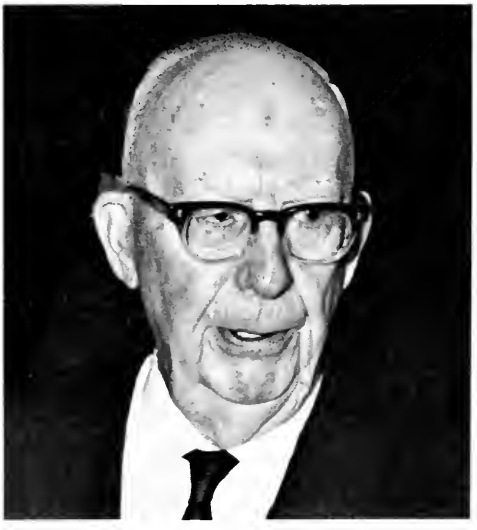
Annual Alumni Banquet

The annual Alumni banquet on Thursday evening at the Lord Baltimore Hotel, attended by more than 600 (a new record) honored the Class of 1918 who received their 50 year diplomas and who recognized the new alumni of the Class of 1968. Guests of honor included: Graduates—Class of 1918; Graduates—Class of 1968; Dr. and Mrs. Thomas B. Turner, Dean, Johns Hopkins University School of Medicine, recipient Medical Alumni Honor Award and Gold Key; Dr. and Mrs. William B. Long, member of the Board of Regents, University of Maryland; Dr. and Mrs. William S. Stone, Dean, University of Maryland School of Medicine, Director of Medical Education and Research; The Rev. Dr. and Mrs. Donald Craig Kerr, Pastor, Roland Park Presbyterian Church; Representative and Mrs. Clarence D. Long, House of Representatives, Washington, D.C.; Mr. J. Lewis Powell, Lecturer and Writer; Dr. and Mrs. William H. Triplett, Executive Director Medical Alumni Association; Dr. and Mrs. Wilfred H. Townshend, Jr., Chairman Alumni Day 1968, Director, Student Health Service, Associate in Medicine, University of Maryland; Dr. and Mrs. John O. Sharrett, President, Medical Alumni Association, Instr. Neurological Surgery, University of Maryland. The meeting presided over by Dr. John O. Sharrett featured Mr. J. Louis Powell who spoke on the subject, "Caveman to Spaceman (The Collapse of Time)." The dinner was concluded with dancing.

Hans R. Wilhelmsen, M.D. '59

L. Zacharian Morgan, M.D. '18
R. Alfred N. Sweet, M.D. '18

Dr. Raymond V. Quinlan



Dr. and Mrs. Harley M. Johnson '18

Mr. Joe Miller, maitre d' Lord Balto. Hotel with son Bruce L. Miller, M.D. '68

ALUMNI BANQUET—LORD BALTIMORE HOTEL

L. Zachariah Morgan, M.D. '18
R. John M. Nicklas, M.D. '18

L. Jos. Sindler, M.D. '18; center, Theo. Kardash, M.D. '42;
r. W. H. Townshend, M.D. '40

L. Morris H. Saffron, M.D. '28
R. Henry Weinert, M.D. '23

L. Thomas B. Turner, M.D. '25
R. Howard B. Mays, M.D. '35

Alumni Physician-Sons Frequent in Class of 1968

At the graduation exercises for the Class of 1968, the sons of 8 alumni of the School of Medicine were graduated in the presence of their fathers, many of whom are not only active in the practice of medicine but who hold important civic and professional posts. Photos taken at the annual Alumni banquet of these father-son combinations are presented.

William J. Fulton, M.D., '22 with son Edwin C. Fulton, M.D. '68

Samuel Legum, M.D., '32 with son Ronald M. Legum, M.D., '68

Frank A. Franklin, M.D., '33 with son Frank A. Franklin, Jr., M.D., '68

Karl Frederick Mech, M.D., '33 with son Karl F. Mech, Jr., M.D. '68

Paul Schonfeld, M.D., '35 with son Burt G. Schonfeld, M.D., '68

William B. Long, M.D. '37 with son William B. Long III, M.D '68

Fuller B. Whitworth, M.D., '39 with son Michael F. Whitworth, M.D., '68

Richard T. Williams, M.D., '40 with son William M. Williams, M.D '68

ALUMNI NEWS REPORT

TO THE BULLETIN:

I would like to report the following: _____

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SUGGESTIONS FOR NEWS ITEMS

American Board Certification
Change of Address
Change of Office
Residency Appointment
Research Completed
News of Another Alumnus
Academic Appointment
Interesting Historic Photographs

Name _____

Address _____

_____

Class _____

Send to

Dr. John A. Wagner, Editor
Bulletin—School of Medicine
University of Maryland
31 S. Greene St.
Baltimore, Md. 21201

BULLETIN *School of Medicine*
University of Maryland

VOLUME 53 OCTOBER, 1968 NUMBER 4

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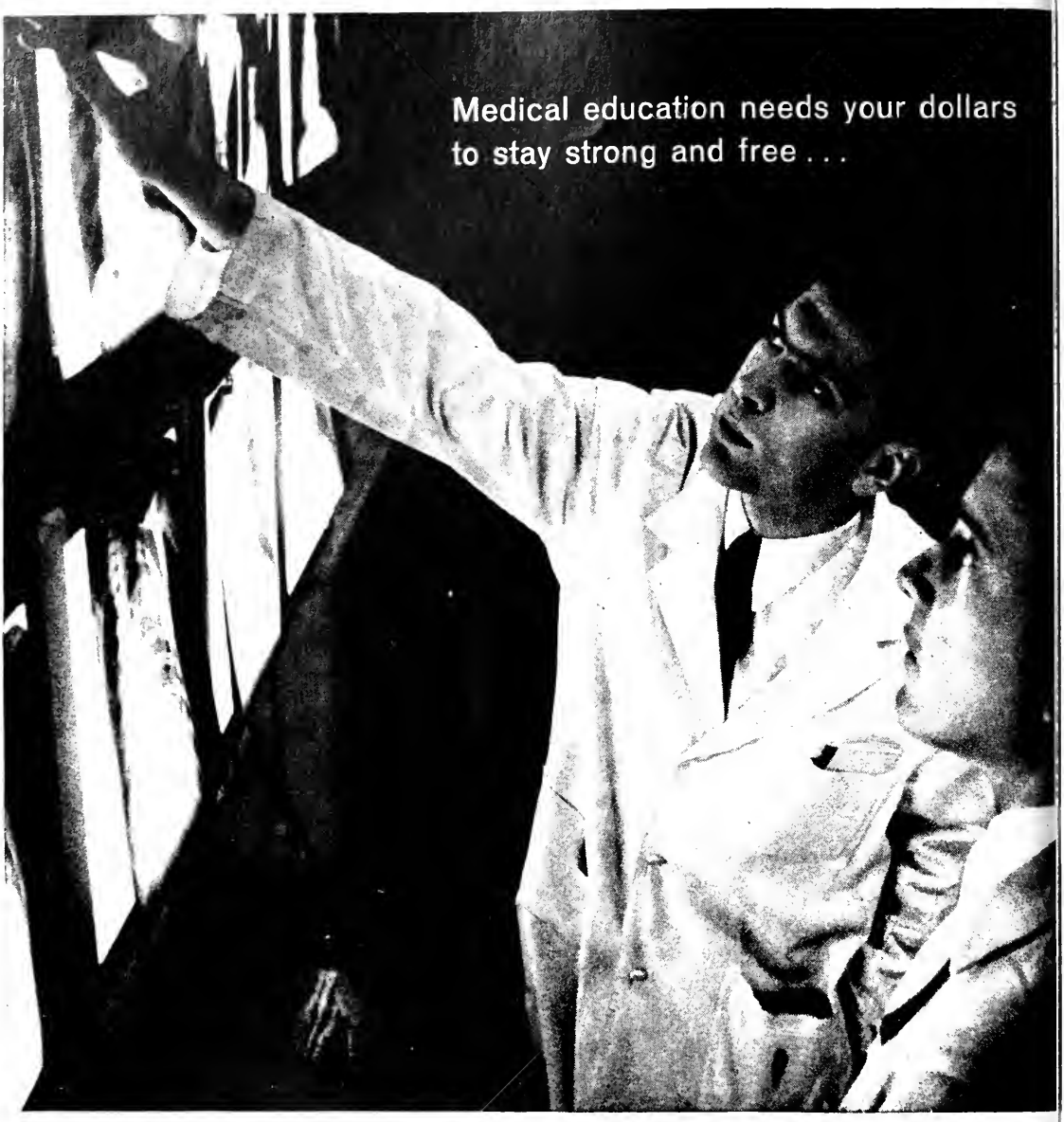

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BULLETIN *School of Medicine*
University of Maryland

VOLUME 53 OCTOBER, 1968 NUMBER 4

Special Notice

AS THIS EDITION of the BULLETIN is being prepared, announcement has been made through the offices of Dr. Wilson H. Elkins, President of the University of Maryland, and Dr. Albin O. Kuhn, Chancellor of the Baltimore Campuses, of the appointment of Dr. John H. Moxley, III, Assistant to the Dean of Harvard University School of Medicine, as Dean of the School of Medicine to succeed Dr. William S. Stone.

Dr. Stone will continue as Dean until the arrival of Dr. Moxley who will begin his tenure on June 1, 1969. A formal announcement of Dr. Moxley's appointment will appear in the next issue of the BULLETIN.

Effect of Magnesium Sulfate on Digoxin-Induced Ventricular Tachycardia in Dogs

by YU-CHEN LEE, M.D.*

In spite of recent advances in the understanding of the mechanism of action and toxicity of digitalis, intoxication to the drug continues to be an important clinical problem. Magnesium sulfate has been reported to be effective in the treatment of arrhythmias produced by digitalis intoxication.[1-7] However, when rapidly administered intravenously it shows only transient effects on digitalis-produced arrhythmias.[1-7] The effects of magnesium sulfate given slowly intravenously over a longer period have not been reported, although it is possible that this method might be more effective.

The beneficial effect of potassium chloride upon digitalis intoxication is acknowledged. The present studies compare the natural course of acute digitalis intoxication with its modification by potassium chloride and magnesium sulfate administration.

Methods

GROUP A (Control). Twenty dogs weighing 8 to 15 kg. were anesthetized intravenously with pentobarbital (30-40 mg./kg.). Lead 2 of the electrocardiogram was monitored on a Sanborn Oscilloscope and permanent records were obtained at appropriate intervals. The femoral artery was cannulated for blood pressure determination and for obtaining blood samples.

Digoxin (0.15 mg/kg.) diluted with 20 cc. of normal saline was given within 3 minutes. If ventricular tachycardia did not develop within 45 minutes after digitalis administration, an additional 0.25 to 0.5 mg. of digoxin was given intravenously.

Blood samples for blood pH, plasma potassium and sodium were obtained before the administration of digoxin, at the onset of ventricular tachycardia and at 30-minute intervals thereafter. The blood pH determination was made with a Leeds-Northrup pH meter, and the sodium and potassium determinations were made with a Baird-Atomic Flame Photometer.

The electrocardiogram was monitored until the return of normal sinus rhythm, or until 4 hours after the development of ventricular tachycardia if sinus rhythm failed to return. The dogs were then transferred to the kennel for 3 days of observation.

GROUP B (Treated with KC1). Twenty dogs were prepared as outlined in Group A. Within 5 minutes after the onset of ventricular tachycardia, KC1 20 mEq. in 250 cc. of normal saline was given by intravenous drip at the rate of 6 to 8 cc. per minute. The rate of KC1 infusion was reduced when sinus rhythm returned, and the infusion was stopped when evidence of severe potassium intoxication appeared. Blood samples were obtained for blood pH, plasma potassium and sodium determination before the administration of digoxin, at the onset of ventricular tachycardia, when sinus rhythm was restored and at 30-minute intervals thereafter. The dogs were observed until at least 2 hours after the restoration of sinus rhythm and were then transferred to the kennel for a 3-day observation period.

* From the Division of Cardiology, Department of Medicine, University of Maryland School of Medicine, Baltimore, Maryland. This investigation was supported by a Maryland Heart Association Research Grant.

GROUP C (Treated with Magnesium Sulfate). Twenty dogs were prepared as described in Group A, but using 5g. of sulfate in 250 cc. of normal saline (2% magnesium sulfate solution), and observations were carried out as in Group B. In addition, plasma magnesium and calcium levels were determined employing an Elmer-Perkins Spectrophotometer.

Results

GROUP A (CONTROL). Ventricular tachycardia developed in each instance, with the onset ranging from 15 to 65 minutes (average 34 minutes). Four dogs died of ventricular fibrillation between 31 and 130 minutes after the onset of ventricular tachycardia. The dogs died during the first night; in one of these dogs, atrial tachycardia had replaced ventricular tachycardia 90 minutes after the onset of ventricular tachycardia. The other dog regained sinus rhythm 210 minutes after the development of ventricular tachycardia. The mortality rate was 30%. In 10 dogs sinus rhythm returned spontaneously 97 to 225 minutes (average 157 minutes) after the onset of ventricular tachycardia. One of these dogs died during the first night. Premature ventricular beats, runs of atrio-ventricular dissociation and marked ST-T changes were common after the return of sinus rhythm. In 5 dogs, all of which survived, ventricular tachycardia lasted more than 4 hours. During the 3-day period of observation, the dogs were weak and anorexic. The plasma sodium was unchanged from the control levels. The plasma potassium showed a slight to moderate increase (0.1 to 1.4 mEq./liter) following digoxin administration. The control blood pH ranged from 7.22 to 7.35 and became 7.34 to 7.51 after digoxin administration as the dogs hyperventilated. There was no significant change in blood pressure among the surviving dogs.

GROUP B (Treated with KC1). The onset of ventricular tachycardia ranged from 15 to 105 minutes (average 35 minutes). Seventeen dogs survived and 3 dogs died (mortality rate 15%). The causes of death for the three dogs were ventricular arrest, ventricular fibrillation, and aspiration, respectively.

In all but one dog sinus rhythm was restored 8 to 70 minutes (average 27 minutes) after KC1 infusion was started. Typical examples of the electrocardiographic changes are shown in Figs. 1 and 2. Following conversion to sinus rhythm, a slow KC1 infusion was required to maintain sinus rhythm. When the rate of KC1 infusion was reduced below a critical level, premature ventricular beats and even ventricular tachycardia reappeared (Fig. 2). An increase in the rate of KC1 infusion usually rapidly abolished the ectopic beats. The electrocardiogram had to be monitored constantly since evidence of severe potassium intoxication could appear suddenly.

The amount of KC1 infusion ranged from 110 cc. to 250 cc. in 19 dogs. In one dog weighing 15 kg., 500 cc. of KC1 were used. Premature ventricular beats were common following the conversion of ventricular tachycardia to sinus rhythm. As in Group A, the dogs were weak and anorectic during the 3-day observation period. The control plasma potassium ranged from 3.2 to 5.4 mEq./liter, and the plasma potassium level at the time of conversion to sinus rhythm ranged from 5.2 to 8.6 mEq./liter (average 6.9 mEq./liter) (Fig. 3). The plasma level of one dog which died of potassium intoxication was 9.2 mEq./liter. The plasma sodium, blood pH and blood pressures were not significantly altered by KC1 infusion.

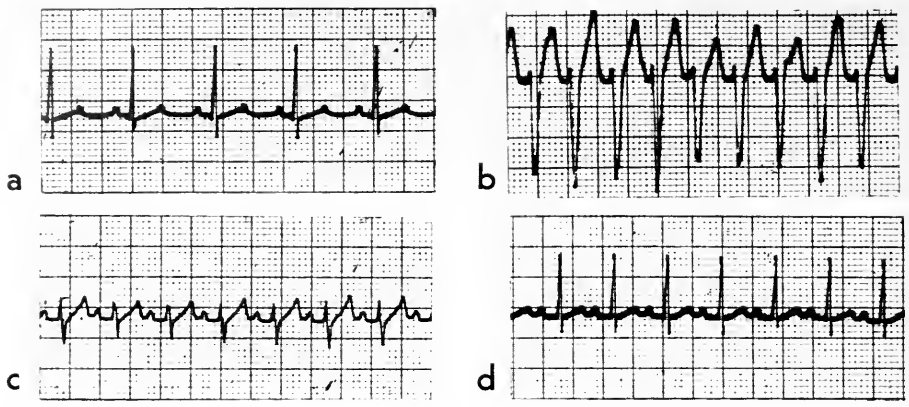

Figure 1. KC1-treated dog (weight 15.0 kg.). a. Control b. Ventricular tachycardia developed 25 minutes after digoxin was given intravenously. c. Sinus rhythm was restored 70 minutes after the initiation of KC1 treatment. There is slight aberration of intraventricular conduction. d. Fifteen minutes later intraventicular conduction became normal, while a slow KC1 drip (0.2 to 0.3 cc. per minute) was continued.

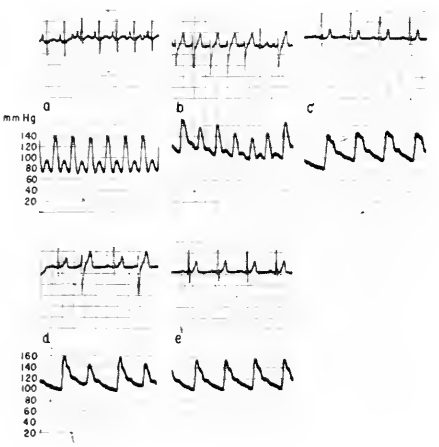

Figure 2. KC-1treated dog (weight 8.0 kg.). a. Control electrocardiogram and femoral arterial pressure (140/72 mmHg). b. Ventricular tachycardia developed 15 minutes after digoxin was given intravenously. There was no drop of blood pressure. c. Sinus rhythm was restored 28 minutes after the initiation of KC1 treatment. d. Ventricular bigeminy developed while slow KC1 drip was given. e. Increase in the rate of KC1 infusion immediately abolished the premature ventricular beats.

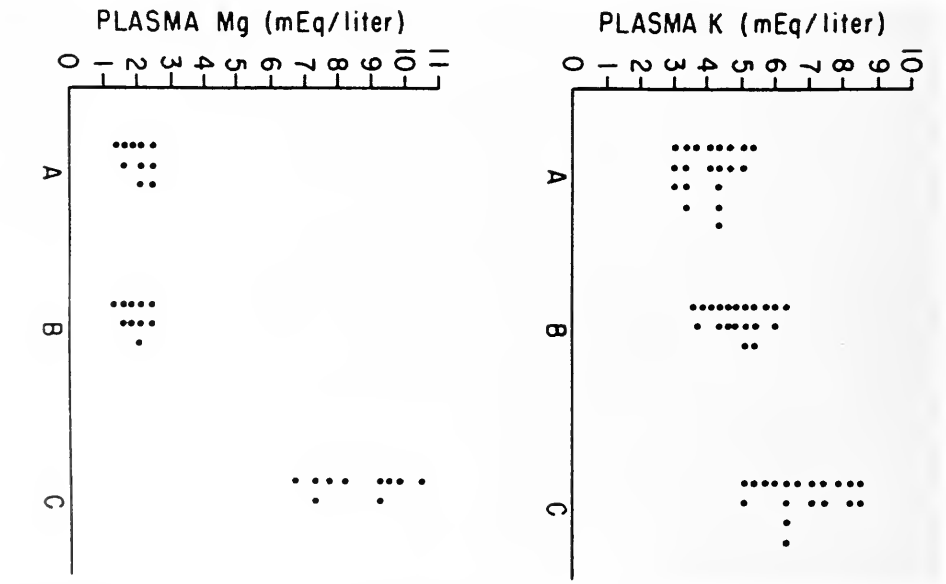

Figure 3. Upper: plasma potassium level in KC1-treated dogs. Lower: plasma magnesium level in magnesium-treated dogs (10 dogs). a. Control. b. Onset of ventricular tachycardia after intravenous digoxin. c. Sinus rhythm restored after treatment with KC1 or magnesium sulfate.

Group C (Treated with Magnesium Sulfate). The onset of ventricular tachycardia after digoxin injection ranged from 16 to 55 minutes (average of 32 minutes). Seventeen dogs survived the 3-day observation period, and 3 dogs died (mortality rate 15%). Sinus rhythm was restored at least temporarily in all 20 dogs 6 to 35 minutes (average 19 minutes) after magnesium sulfate infusion was started. Two of these dogs manifested a basic normal sinus rhythm with ventricular bigeminy. Marked prolongation of the PR interval was noted in 12 dogs after conversion to sinus rhythm. The maintenance of sinus rhythm was more difficult than in the KC1-treated group. The typical electrocardiographic changes are shown in Figs. 4 and 5.

Ventricular tachycardia returned in each instance if the magnesium sulfate infusion was stopped shortly after the conversion to sinus rhythm. Therefore, magnesium sulfate was infused continuously in every instance. The required rate of magnesium sulfate infusion varied from 0.4 to 3.6 cc. per minute, with the total amount infused being 190 to 250 cc. The rate of magnesium sulfate infusion was reduced gradually if sinus rhythm continued. After the cessation of magnesium sulfate infusion premature ventricular beats appeared in all instances.

In two dogs in which sinus rhythm appeared 12 and 30 minutes, respectively, after magnesium sulfate infusion was started, sinus rhythm lasted only temporarily despite continuous magnesium sulfate infusion. In one dog sinus rhythm was restored 15 minutes after magnesium sulfate was started, and the electrocardiogram continued to show sinus rhythm at the end of the period of observation. However, the dog died during the first night.

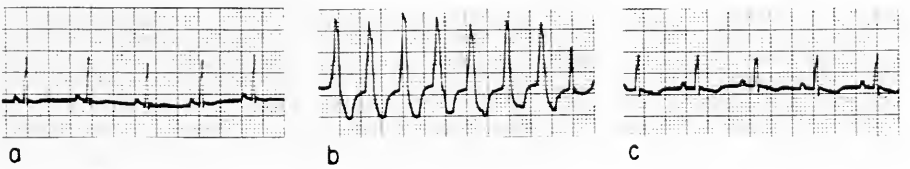

Figure 4. Magnesium sulfate-treated dog (weight 12.8 kg.). a. Control. b. Ventricular tachycardia developed 38 minutes after digoxin was given intravenously. c. Sinus rhythm was restored 24 minutes after the initiation of the magnesium sulfate treatment.

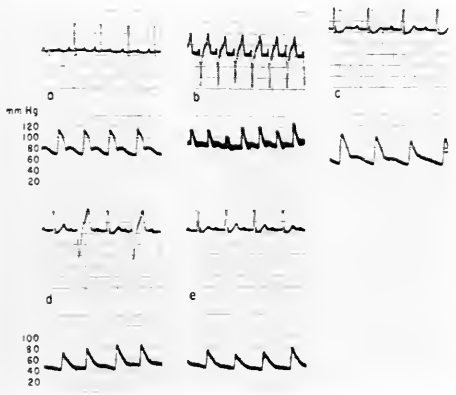

Figure 5. Magnesium sulfate-treated dog (weight 12.1 kg.). a. Control electrocardiogram and femoral arterial pressure (116/68 mmHg). b. Ventricuuar tachycardia developed 52 minutes after digoxin was given intravenously. Blood pressure was 120/80 mmHg. c. Sinus rhythm with first degree atrio-ventricular block developed 32 minutes after the initiation of the magnesium sulfate treatment. PR 0.20 seconds. Blood pressure 100/48 mmHg. d. Ventricular bigeminy developed when magnesium sulfate infusion was slowed down to 1 cc. per minute. Blood pressure 86/46 mmHg. e. Premature ventricular beats were abolished within 2 minutes after the rate of magnesium infusion was increased to 2 cc. per minute.

In one dog sinus rhythm was restored 7 minutes after the magnesium sulfate infusion was started; ventricular tachycardia returned when the rate of magnesium sulfate infusion was decreased. An increase in the rate of magnesium sulfate infusion resulted in marked sinus bradycardia with a prolonged PR interval. The magnesium sulfate infusion was therefore stopped. Ventricular tachycardia returned in 6 minutes, and the dog died of ventricular fibrillation.

In one dog sinus rhythm was restored 8 minutes after the magnesium sulfate infusion was started. When the rate of magnesium sulfate was decreased, ventricular tachycardia reappeared. When the rate of magnesium sulfate infusion was increased, the dog developed ventricular arrest and died (Fig. 6).

Magnesium sulfate produced a moderate drop in blood pressure in every instance; i.e., 15 to 35 mmHg decrease in systolic and 10 to 30 mmHg decrease in diastolic pressure. Plasma magnesium and calcium levels were determined in 10 dogs. The control plasma magnesium level ranged from 1.5 to 2.7 mEq./liter and no significant changes were noted following digitalis injection. The plasma magnesium level at the time of conversion to sinus rhythm following magnesium sulfate infusion ranged from 6.8 to 10.6 mEq./liter. (Fig. 3). Plasma calcium showed no significant change in 7 dogs and was slightly decreased (0.6 to 1.1 mg. percentum), in 3 dogs following magnesium sulfate infusion. Plasma potassium and sodium remained unchanged after magnesium sulfate infusion.

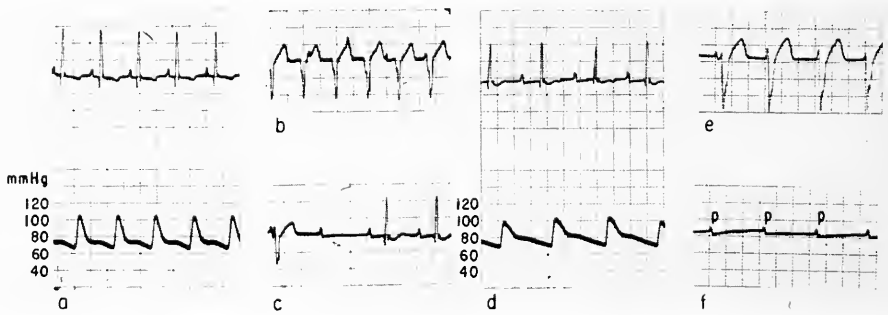

Figure 6. Magnesium sulfate-treated dog (weight 8.2 kg.). a. Control electrocardiogram and femoral arterial pressure (108/64 mmHg). b. Ventricular tachycardia developed 20 minutes after digoxin was given intravenously. c. Nineteen minutes after the institution of magnesium sulfate treatment, ventricular tachycardia was abolished and sinus rhythm was restored. The PR interval is markedly prolonged. There is transient second degree atrio-ventricular block. d. Sinus rhythm with first degree atrio-ventricular block was maintained while a slow magnesium sulfate drip (1.5 cc. per minute) was continued. Blood pressure 100/64 mmHg. e. Twenty-one minutes after restoration of sinus rhythm, ventricular tachycardia reappeared despite continuation of magnesium sulfate drip at a rate of 1.5 cc. per minute. f. The rate of magnesium sulfate infusion was increased to 5 cc. per minute. Twelve minutes later, ventricular standstill developed and the dog died.

Comment

The pharmacological actions of parenterally administered magnesium salts have been extensively studied by many investigators. The magnesium ion is known to have profound effects on the cardiovascular system including marked vasodilation,[8, 9] a fall in blood pressure,[8, 10] inhibitory effects on all parts of the conduction system and probably some direct toxic action on the myocardium itself.[11] The magnesium ion was first utilized for the treatment of digitalis-induced arrhythmia by Zwillinger[1] who reported striking success in abolishing extrasystoles caused by digitalis intoxication. However, the duration of this effect was temporary and this observation was confirmed by subsequent investigators.[5-7] Rothberger and Zwillinger[2] demonstrated

the effectiveness of magnesium in abolishing ventricular tachycardia induced by strophanthin or barium in dogs.

Freundlich[5] reported a case of bi-directional ventricular tachycardia owing to digitalis which was temporarily abolished by magnesium. Enselberg, *et al.*,[6] gave magnesium sulfate intravenously to 25 patients with a variety of arrhythmias including 13 owing to digitalis intoxication. The ventricular extrasystoles, whether due to digitalis or other causes, were abolished or sharply reduced in frequency. In some instances there was a transient increase in the frequency at the beginning or end of magnesium administration. The cardiac effects were rapid in onset and of very brief duration. It was thus concluded that the therapeutic use of magnesium in arrhythmias is limited by its ephemeral action and by its occasional undesirable effect. The mechanism by which magnesium sulfate counteracts digitalis induced arrhythmias is probably due to its nonspecific inhibitory effect on cardiac irritability.

The method employed by previous investigators was the rapid intravenous injection of 10 to 30 cc. of 10 to 20% magnesium sulfate solution.

In the present studies 2% magnesium sulfate solution was given by intravenous drip at the rate of 6 to 8 cc. per minute. In all dogs sinus rhythm was restored, at least temporarily, 6 to 35 minutes after the beginning of magnesium sulfate infusion. The ability of magnesium sulfate to maintain sinus rhythm varied widely among the treated dogs. In 2 dogs sinus rhythm lasted only for a few minutes, and ventricular tachycardia returned despite the continuous infusion of magnesium sulfate. However, sinus rhythm was maintained with relatively small amounts of magnesium sulfate in 8 additional dogs. When the rate of magnesium sulfate infusion was decreased below a critical level, premature ventricular beats or ventricular tachycardia returned. An increase in the rate of magnesium sulfate infusion was usually followed by the restoration of sinus rhythm.

Magnesium sulfate infusion more rapidly converted ventricular tachycardia to sinus rhythm than did KC1; the average for magnesium sulfate was 19 minutes, whereas for KC1 it was 27 minutes. Treatment with either of these drugs reduced the mortality to 15%, whereas the mortality in controls was 30%.

However, magnesium sulfate caused a moderate hypotension and also produced a high degree of atrio-ventricular block in some of the dogs. These effects were not seen in the KC1-treated dogs.

Because of these serious side effects, magnesium sulfate cannot be recommended for the routine treatment of digitalis-induced ventricular tachycardia.

Summary

Digoxin was given intravenously to produce ventricular tachycardia in 60 dogs. Twenty dogs were utilized as controls. Among the control dogs, 30% died. In 10 dogs, sinus rhythm returned spontaneously 97 to 225 minutes (average 157 minutes) after the onset of ventricular tachycardia.

Twenty dogs were treated with an intravenous drip of KC1 20 mEq. in 250 cc. of normal saline at the rate of 6 to 8 cc. per minute. Sinus rhythm was restored in 19 dogs 8 to 70 minutes (average 27 minutes) after KC1 infusion was started. Fifteen percent of the KC1-treated dogs died.

Twenty dogs were treated with an intravenous drip of 2% magnesium sulfate in 250 cc. of normal saline at a rate of 6 to 8 cc. per minute. Sinus rhythm

was restored at least temporarily in all 20 dogs 6 to 35 minutes (average 19 minutes) after magnesium sulfate infusion was started. The maintenance of sinus rhythm was more difficult as compared with the KC1-treated dogs. Fifteen percent of the magnesium-treated dogs died. Magnesium may produce a profound hypotensive effect and marked atrio-ventricular block. These side effects were not seen in KC1-treated dogs.

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[2] ROTHBERGER, C. J., AND ZWILLINGER, L.: Uber die wirkung von magnesium auf die strophanthin—und die barium-tachykardie. *Arch. Exp. Path. Pharmakol., 181:*301, 1936.

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MEDICAL SCHOOL SECTION

Dean's LETTER

Dear Students, Medical Alumni and Friends of the Medical School:

There is much talk these days about the development of new and innovative ways of instruction that will revolutionize education. It is always worthwhile to question, to evaluate and to act as far as possible on factual information backed by substantial evidence. If new and innovative ideas and approaches to learning are so tested they may lead to real improvement. However, if this type of objective evaluation is not used, new and innovating ideas may be very misleading. For example in teaching children to read, it has been advocated by a considerable group of educators that the alphabetical phonetic approach to reading be discarded and word association with objects and actions substituted for it.

A scientific evaluation of research on methods used in teaching primary students to read from 1912 to 1965 conducted by Dr. Jeanne S. Chall, Professor of Education at Harvard in 1967, and reported in the issue of the *Carnegie Quarterly* for summer 1967 reveals that regardless of the background of the child and the ability to learn, the alphabetical phonetic code emphasis approach to learning to read, long used in American education programs for primary students, is substantially better than any other method tried.

For similar reasons the medical curricula and past methods of teaching are not being abandoned for untried ideas and methods. New ideas are being tried in the Medical School but they are not being adopted until there is substantial evidence to support the need for change.

Evidence today supports the Medical School experience that an excellent background in the arts and science is the best preparation for the study of medicine. The medical curricula should present a core of knowledge in the basic sciences and clinical subjects that will provide a sound foundation upon which can be developed, in the intern and residency periods, the skills and judgment required for the practice of medicine.

Sincerely,

WILLIAM S. STONE, M.D.
Dean

Faculty NOTES

Dr. Frank H. J. Figge was awarded the degree of Doctor of Science *Honoris Causa* in May, 1968, by his alma mater, Colorado College, Colorado Springs, Colorado. The citation commended Dr. Figge for his many contributions to medical science and medical education.

Drs. Arlie Mansberger, Carroll Spurling and **Kurt Sligar** have been chosen by the Senior and Sophomore classes of the School of Medicine to receive the 1968 Student American Medical Association Golden Apple Award for excellency in teaching. **Dr. Mansberger,** Associate Professor of Surgery, was honored for outstanding service on the clinical staff; **Dr. Spurling,** Associate Professor of Medicine, for preclinical service; and **Dr. Sligar,** a medical resident, for his efforts on the house staff.

Dr. Crosby Authors Book

Dr. Robert M. N. Crosby, Assistant Professor of Neurosurgery, is the author of a volume entitled, *The Waysiders.* The book concerns the existence, possible causes and the treatment of dyslexia, and was published in collaboration with Robert A. Liston.

Dr. Albert F. Heck, Associate Professor of Neurology, traveled to Finland this summer at the invitation of the Finnish Neurosurgical Society to speak on "Opacity Pulse Propagation in the Cerebral Cortex of Experimental Animals." He then gave a lecture on heredoataxias to staff members and students at the University of Stockholm. Dr. Heck also attended the Fifth European Conference on Microcirculation in Gothenberg, Sweden, where he presented a paper entitled "Opacity Pulse and Pulse Propagation in the Microcirculation; Physiologic Reactance of a New Technique." Miss Ilse Hawthorne, an investigator in the Shock-Trauma Program of the Department of Surgery, was co-author of the paper.

Dr. Heck is presently engaged in the investigation of the neural control of cardiovascular function and vasomotor mechanisms in the Department of Neurology and in the Shock-Trauma Unit to which he is the neurological consultant.

Dr. Robert L. Derbyshire, Associate Professor of Sociology in Psychiatry and Director of Undergraduate Psychiatric Education, received the Seventh Annual Student Council Faculty Man of the Year award for 1968.

Dr. Brody Made Member Advisory Board at Peruvian University

Dr. Eugene B. Brody, director of the Psychiatric Institute of the School of Medicine, while in Lima, Peru, recently was installed as a member of the International Scientific Advisory Board of the newly established Institute of Social Psychiatry of the University of San Marcos.

Retired Professor Still Active

Ruth D. Musser, who before her retirement in 1966, was assistant professor of pharmacology at the University of Maryland School of Medicine and chairman of pharmacology at the school of nursing, has been elected treasurer of the National League of American Pen Women.

The fourth edition of Mrs. Musser's textbook *Pharmacology and Therapeutics* is now in press with Dr. John J. O'Neill, associate professor of cell biology and pharmacology at the medical school, as the new co-author. Since its first publication in 1959 it has proved to be one of Macmillan's best-selling textbooks.

Mrs. Musser also collaborated with her colleagues at the medical school in more than 20 research papers published in scientific journals.

1968 Class Receive Diplomas and Announce Where They Will Serve Internships

On June 8th at 10:30 in the morning, the Class of 1968 received their degrees as Doctors of medicine and departed for College Park Campus to receive their diplomas and enter upon their several careers as physicians. Members of the Class of 1968 will serve internships in the following institutions:

ALLISON, SAMUEL B.
Geisinger Medical Center, Danville, Pa.

AMOSS, WILLARD P.
Public Health Service Hospital, Baltimore.

BAUM, RICHARD A.
George Washington Hospital, Washington, D. C.

BEAMON, CHARLES R., JR.
Presbyterian Hospital, New York, N. Y.

BEARMAN, SHELDON B.
Mount Sinai Hospital, New York, N. Y.

BENESON, MICHAEL W.
Ben Taub-V. A. Hospital, Houston, Tex.

BLUM, BARRY A.
South Baltimore General Hospital, Baltimore.

BLUMBERG, MORTON B.
Santa Barbara Cottage, Santa Barbara, Calif.

BOWEN, BRUCE J.
Memorial Hospital of Long Beach, Long Beach, Calif.

BRITTON, ROBERT M.
Memorial Hospital of Long Beach, Long Beach, Calif.

BRULL, ROBERT
Sinai Hospital of Baltimore, Baltimore, Md.

BUDDINGTON, RICHARD S.
Duke University Hospital, Durham, N. C.

CALDWELL, JOHN L.
Washington Hospital Center, Washington.

CALLAGHAN, JOSEPH
Rhode Island Hospital, Providence, R. I.

CAPLAN, ELLIS S.
University Hospital, Baltimore, Md.

CHERRY, JOEL M.
University Hospital, Baltimore, Md.

CLOPPER, TODD D.
Washington Hospital Center, Washington.

COHEN, ELLIOT S.
St. Francis Hospital, Honolulu, Hawaii

COLLIGAN, FRANKLIN W.
South Baltimore General Hospital, Baltimore.

DAW, ALBERT LEE
South Baltimore General Hospital, Baltimore.

DEEGAN, MICHAEL J.
University Hospital, Baltimore, Md.

EDWARDS, CHARLES C.
Yale New Haven Medical Center, New Haven, Conn.

EGLOFF, ALLEN C.
Jefferson Hospital, Jefferson Medical College, Philadelphia, Pa.

FAUSEL, ROBERT W., JR.
Conemaugh Valley Memorial Hospital, Johnstown, Pa.

FELDMAN, GERALD B.
Sinai Hospital, Baltimore, Md.

FLIGSTEN, KENNETH E.
Baltimore City Hospitals, Baltimore, Md.

FRANKLIN, FRANK A., JR.
St. Louis Children's Hospital, St. Louis, Mo.

FRIEDMAN, HOWARD R.
Sinai Hospital, Baltimore, Md.

FRIZZERA, JOHN G.
South Baltimore General Hospital, Baltimore.

FULTON, EDWIN C.
George Washington General Hospital (D. C. General Hospital), Washington, D. C.

GAMBRILL, RAYMOND, III
South Baltimore General Hospital, Baltimore.

GEHLERT, SIDNEY R., III
South Baltimore General Hospital, Baltimore.

GELIN, JOHN D.
Public Health Service Hospital, Baltimore.

GLICK, RONALD S.
Kaiser Foundation Hospital, San Francisco, Calif.

GOLDSTEIN, WILLIAM N.
Washington Hospital Center, Washington.

GREEN, GERALD I.
Washington Hospital Center, Washington.

GROOVER, JACK R.
Washington Hospital Center, Washington.

HANDWERGER, BARRY S.
Mount Sinai Hospital, New York, N. Y.

HARRIS, ROGER C.
Washington Hospital Center, Washington.

HERMAN, MELVIN H., JR.
Army Medical Service Hospital (Walter Reed Hospital), Washington, D. C.

HESS, DOUGLAS B.
York Hospital, York, Pa.

HOOPER, STEPHEN L.
York Hospital, York, Pa.

HOROWITZ, IRVIN R.
Medical College of Virginia, Richmond, Va.

HYMAN, GEORGE F.
Washington Hospital Center, Washington.

KANE, JAMES G., JR.
St. Agnes Hospital, Baltimore, Md.

KEECH, RICHARD C.
Maryland General Hospital (University of Maryland Hospital), Baltimore, Md.

KEEGAN, KIRK A., JR.
U.S. Air Force, Grant Air Force Hospital, Travis A.F.B., Calif.

KNEFELY, GEORGE M., JR.
Army Medical Service Hospitals, Tripler Army Hospital, Honolulu, Hawaii

KNOWLES, FREDERICK E.
University Hospital, Baltimore, Md.

KOSKI, CAROL L.
University Hospital, Baltimore, Md.

KULIK, FRANK A.
South Baltimore General Hospital, Baltimore.

LANCELOTTA, CHARLES J.
St. Agnes Hospital, Baltimore, Md.

LAZARUS, BARRY A.
Mount Zion Hospital, San Francisco, Calif.

LEGUM, RONALD M.
Union Memorial Hospital, Baltimore, Md.

LEVENSON, STANLEY M.
Washington Hospital Center, Washington.

LEVIN, GORDON L.
Highland General Hospital, Oakland, Calif.

LITT, ABRAHAM A.
Passavant Memorial Hospital, Chicago, Ill.

LITTLE, RAYMOND R.
University Hospital, Baltimore, Maryland

LITTMAN, PHILIP
Washington Hospital Center, Washington.

LONG, WILLIAM B., III
University Hospital, Baltimore, Md.

MAHONEY, CARROLL D.
Washington Hospital Center, Washington.

MALINOW, STANFORD H.
Sinai Hospital, Baltimore, Maryland

MANEKIN, STEVEN F.
Sinai Hospital, Baltimore, Maryland

MCGUIRE, TERRANCE A.
Washington Hospital Center, Washington.

MCNINCH, EUGENE R., JR.
Conemaugh Valley Memorial Hospital, Johnstown, Pa.

MECH, KARL F., JR.
Presbyterian Hospital, Pittsburgh, Pa.

MENDELSOHN, HERBERT E.
South Baltimore General Hospital, Baltimore.

MERLIS, ANTHONY L.
Passavant Memorial Hospital, Chicago, Ill.

MIKESELL, KATHRYN A.
University Hospital, Baltimore, Md.

MILLER, BRUCE L.
Harbor General Hospital, Torrance, Calif.

MORGAN, BEVERLY E. J.
Newark Beth Israel Hospital, Newark, N. J.

MORTON, BERT F.
St. Agnes Hospital, Baltimore, Md.

NORDGREN, A. CURTIS
McGill University, Royal Victoria Hospital, Montreal, Canada

NORWOOD, THOMAS H.
University of Washington Hospitals, Seattle, Wash.

PATTEE, BURTON C.
Denver General Hospital, Denver, Colo.

POTOTSKY, RONALD S.
University Hospital, Baltimore, Maryland

QUILLEN, CARL G.
Jackson Memorial Hospital, Miami, Fla.

RANKIN, THOMAS V.
Charity-Tulane Division, New Orleans, La.

REED, WILLIAM A.
Kaiser Foundation, Oakland, Calif.

RENBAUM, JOEL W.
Washington Hospital Center, Washington.

RIDDLESBERGER, MERCHELINE
Kaiser Foundation, Oakland, California

RILEY, DAVID J.
Baltimore City Hospitals, Baltimore, Md.

RIMASH, RORICK T.
Albany Hospital, Albany Medical Center, Albany, New York

RIVERA, LUIS R.
Beth Israel Hospital, New York, New York

ROIHL, NORBERT H.
Greenwich Hospital, Greenwich, Connecticut

ROSENBAUM, STEPHEN D.
Sinai Hospital, Baltimore, Md.

ROSENSTEEL, ROBERT J.
South Baltimore General Hospital, Baltimore, Md.

ROSENSTOCK, JEFFREY G.
Bellevue Hospital, New York University, New York, N. Y.

SAMORODIN, CHARLES S.
University Hospital, Baltimore, Md.

SCHAEFFER, WALTER C.
Naval Hospitals, Philadelphia, Pa.

SCHLOSSBERG, BARRY J.
University Hospital, Baltimore, Md.

SCHONFELD, BURT G.
Public Health Service Hospital, San Francisco, Calif.

SEMINS, HOWARD
Mercy Hospital, Pittsburgh, Pa.

SHACK, MICHAEL J.
Colorado Medical Center, Denver, Colo.

SHAW, JOHN M.
Passavant Memorial Hospital, Chicago, Ill.

SIEGEL, ETHEL *To be announced*

SPIELMAN, STUART H.
Sinai Hospital, Baltimore, Md.

STAFFORD, JOHN D.
Santa Barbara Cottage Hospital, Santa Barbara, Calif.

STAUFER, WILFRED B.
Conemaugh Valley Memorial Hospital, Johnstown, Pa.

STUART, FRANKLIN R.
Washington Hospital Center, Washington.

STUPPLER, STEPHEN A.
Public Health Service Hospital, Seattle, Wash.

TANNENBAUM, ALICE S.
Bellevue Hospital, New York University, New York, N. Y.

TURNER, ELIZABETH A.
York Hospital, York, Pa.

VALIGORSKY, JON M.
University Hospital, Baltimore, Md.

VERGNE-MARINI, PEDRO
University of Puerto Rico, Rio Piedras, Puerto Rico (University District Hospital)

VOLCJAK, EDWARD E.
Naval Hospitals, Philadelphia, Pa.

WEIMER, STANLEY R.
South Baltimore General Hospital, Baltimore, Md.

WELSH, JAMES J., JR.
Army Medical Service Hospitals, Brooke Hospital, San Antonio, Tex.

WHITWORTH, MICHAEL F.
Orange County General Hospital, Orange County, California

WIDMEYER, ROBERT S., II
St. Agnes Hospital, Baltimore, Md.

WILLIAMS, ROBERT T.
Highland Hospital, Rochester, N. Y.

WILLIAMS, WILLIAM M.
St. Luke's Hospital, Denver, Colo.

WILLIS, EUGENE, JR.
Georgetown D. C. General Hospital, Washington D. C. (D. C. General Hospital)

WINAKUR, STUART
University Hospitals, Columbus, Ohio

WINTER, STEPHEN L.
Maryland General Hospital (University of Maryland Hospital), Baltimore, Md.

WOLFE, IRVING D.
Public Health Service Hospital, Baltimore.

WOODROW, KENNETH M.
Kaiser Foundation, Oakland, Calif.

YOUNG, EDWARD J.
Washington Hospital Center, Washington.

Committee Appointed to Discuss Merger of Cheverly Hospital with University of Maryland Hospital

At the end of the academic year, Dr. William S. Stone, Dean, appointed a committee to merge with a similar group from the Prince George's County Hospital at Cheverly to discuss the actual procedure for an affiliation program between the hospital and the School of Medicine. The committee is charged with development of a number of important facets which should lead to an active program of affiliation for medical students as well as house officers, should the arrangements be completed. Dr. Eugene J. Linberg is Chairman of the committee which includes Drs. Woodward, Buxton, Haskins, Dennis, Weaver and Schultz.

Walter Dent Wise

1885-1968

On July 23, 1968 one of the most distinguished and beloved graduates of the University of Maryland Medical School (College of Physicians and Surgeons, 1906) died. He will live in the memory of his friends as a lovable person of sterling character and as a surgeon who possessed a swift sureness in approach and a gentleness of touch.

Walter Dent Wise was born on May 18, 1885 in Patuxent Beach, Maryland, the son of Walter Hanson Briscoe Stone Wise and Martha Jane Dent.

Following graduation from Medical School he trained in Surgery at the Baltimore City Hospitals and Mercy Hospital, Baltimore, Maryland.

He was appointed Chief Surgeon of Mercy Hospital in 1935, a position that he held until June 1955.

He was Professor of Clinical Surgery, University of Maryland Medical School from 1932 to 1937 and Professor of Surgery from 1937 to 1955, following which he was given the title of Emeritus Professor.

He was appointed a lecturer in Surgery at the Johns Hopkins Medical School in 1947.

During World War II he was Medical Director of the Selective Service of Maryland from April 1942 to August 1943. He then served as Consultant to the Third Medical Service Command with the rank of Colonel. He was awarded the Legion of Merit, December 12, 1945.

In 1961 he received the honor award and gold key of the Medical Alumni Association in the University of Maryland. He served as President of the Medical Alumni Association from 1942 to 1943.

Professional recognition was widespread.

He was Secretary of the Medical and Chirurgical Faculty of the State of Maryland from 1932 to 1939, and Chairman of the Council of that Association from 1947 to 1951, and was elected to the Presidency in 1951.

He was Vice-President of the Baltimore City Medical Society from 1941 to 1942 and President from 1942 to 1943.

He was President of the Board of Governors of Mercy Hospital from 1937 to 1961 and also served as Chairman of the Executive Committee of that hospital.

He was Chairman of the Maryland Hospital Survey Committee from 1947

to 1948 and Chairman of the Advisory Council of Hospital Construction of the State Board of Health from 1948 to 1962, when he resigned.

He was Co-Chairman of the Mercy Hospital Building Committee from 1960 until his death.

He held membership in the following professional associations:

Baltimore City Medical Society
Medical and Chirurgical Faculty of the State of Maryland
American Medical Association
American College of Surgeons
American Surgical Association
Southern Surgical Association
American Association for the Surgery of Trauma
Founders Group—American Board of Surgery
Society of Medical Consultants to the Armed Forces

He was a member of the Maryland Club and served as its President from 1949 to 1954.

He was also a member of the Elkridge Club.

His first wife, Agnes Gordon Whiting Wise, died in 1919 and his second wife, Josephine Warfield McMillan Wise, died in 1954. Two daughters, by the first marriage, are deceased.

His dedicated efforts established great respect for him. All who knew him are deeply saddened at his passing.

GEORGE H. YEAGER, M.D.

English Student First Recipient of New Pathology Fellowship

Dr. Lester Kiefer, Director of Laboratories at the Memorial Hospital in Cumberland, Maryland, has donated to the Department of Pathology of the School of Medicine, a student fellowship to be awarded annually to an undergraduate student who wishes to undertake special studies in the Department of Pathology.

In accepting the fellowship in behalf of the Department of Pathology, Dr. Robert B. Schultz, Professor of Pathology, commented on the great value of this fellowship in enabling promising young men to further their ambitions in the study of pathology, most appropriately during the summer season. The first recipient of the annual Kiefer Student Fellowship is Mr. Robert Fields, an upperclassman at the Middlesex Hospital School of Medicine in London.

Dr. Kiefer, an alumnus of the University of Pennsylvania School of Medicine and a former Chief Resident in Pathology at the University of Maryland, also serves as Visiting Assistant Professor in Pathology at the School of Medicine. He is currently President of the Allegheny Medical Society and is active as a member of the Maryland Division of the American Cancer Society, serving on a number of important committees.

Maryland Society for Medical Research Acquires New Film

The Maryland Society for Medical Research recently purchased "Safe Handling of Laboratory Animals," a 14-minute color film produced under the auspices of the Public Health Service and filmed at the animal quarters of the National Institute of Health in Bethesda.

In the film proper procedures are demonstrated for cleaning, feeding, watering, bedding, picking up, and handling laboratory animals such as rabbits, mice, hamsters, dogs, cats, and monkeys. The film also shows precautionary measures to be taken by laboratory personnel to protect themselves from disease or injury. The important role played by animal experimentation in conquering polio, diptheria, rabies, etc. is also stressed.

Group may obtain the film on a free loan basis through the Society's office at 522 West Lombard Street, Baltimore, Maryland, or by calling MU 5-5348.

International Units Of Measure

Scientific publications, hospital charts and laboratory reports are often noted for the wide variations in symbols and abbreviations. A general conference on weights and measures was held in Paris during the month of October, 1967. The BULLETIN takes pleasure in reproducing the recommendations of this conference.

Changes Recently Adopted

At the 13th General Conference on Weights and Measures, in Paris, October 13, 1967, the following actions were taken with reference to changes in definition, designation, or abbreviation of certain units of measure (*Technical News Bulletin* of National Bureau of Standards, Vol. 52, January 1968).

The National Bureau of Standards uses the International System (SI) of Units for designation of the following 6 basic quantities:

Quantity	*Unit*	*Symbol*
Length	meter	m
Mass	kilogram	kg
Time	second	s
Electric current	ampere	A
Thermodynamic temperature	degree Kelvin	°K
Luminous intensity	candela	cd

Derived Units of Measurement

Quantity	*Derived Unit*	*Symbol*
Area	square meter	m^2
Volume	cubic	m^3
Density	kilograms per cubic meter	Kg/m^3
Velocity	meters per second	m/s
Force	newton	$N(kg \cdot m/s^2)$
Pressure	newton per sq. meter	N/m^2
Work, energy, quantity of heat	joule	J (N.m)
Power	watt	W (J/s)
Electric charge	coulomb	C (A·s)
Voltage, potential difference, electromotive force	volt	V (W/A)
Electric field strength	volt per meter	V/m
Electric resistance	ohm	V/A
Electric capacitance	farad	F (A·s/V)
Magnetomotive force	ampere	A
Luminance	candela per sq. meter	cd/m^2

Time

A new definition of the international unit of time, the *second* ("atomic second") was adopted, based upon a characteristic rate of electromagnetic oscillation of the cesium-133 atom.

Length

The name, "micron," for a unit of length equal to 10^{-6} meter, and the symbol "μ", by which it has been indicated, were abandoned. The symbol "μ" is to be used solely as an abbreviation for the prefix "micro-," standing for multiplication by 10^{-6}. Thus, the length previously designated as micron (μ) should be designated micrometer (μm).

X-radiation

The General Conference on Weights and Measures has also authorized use of the symbol, R (instead of r), for roentgens.

Units, Prefixes, and Symbols

According to the *1965 Technical Conference Transactions* (p. 437), the names of multiples and submultiples of the units and their prefixes and symbols are as follows:

Factor by Which Unit is Multipleid	*Prefix*	*Symbol*
10^{12}	tera	T
10^{9}	giga	G
10^{6}	mega	M
10^{3}	kilo	k
10^{2}	hecto	h
10^{1}	deca	da
10^{-1}	deci	d
10^{-2}	centi	c
10^{-3}	milli	m
10^{-6}	micro	μ
10^{-9}	nano	n
10^{-12}	pico	p
10^{-15}	femto	f
10^{-18}	atto	a

The BULLETIN—*A Reappraisal*

EVERY LONG-STANDING institution should periodically undergo the test of an inspection and reappraisal of its accomplishments, its policies and its objectives. Accordingly, early in the spring of 1968, the Dean of the School of Medicine appointed the faculty committee to investigate the status of the BULLETIN of the School of Medicine and to make recommendations to the faculty relative to its continuance.

Headed by Dr. Robert B. Schultz, this committee met on February 14, 1968. The committee's report was presented to the faculty board and adopted at its meeting on May 22. The report is as follows:

1. *The* BULLETIN *should activate school interest in the eyes of the alumni.*

To the end stated above, the BULLETIN should contain reviews concerning matters of importance to the progress of the School of Medicine, such as: the progress of curriculum changes and the forces motivating them; current building programs and programs of revisions in the physical plant; position papers concerning matters of concern to faculty and alumni alike, such as the current budget "crisis"; the role of the School of Medicine in the Regional Medical Program, including progress reports, the interrelations between school and alumni, etc.; postgraduate education, its offerings and aspiration.

2. *The* BULLETIN *should serve an alumni function.*

By the above is meant that matters of concern to alumni, apart from affairs of the school, should be offered in the BULLETIN.

3. *The* BULLETIN *should offer scientific papers as follows:*
   a. Student papers of a meritorious nature
   b. Faculty papers as follows:
      1) "Review" papers by faculty, especially those that are clinically oriented
      2) Preliminary reports of work with larger programs underway
      3) Those meritorious papers for which the BULLETIN would be an "alternate" to the "ideal" national journal
      4) Historical papers

In connection with the latter, it is noted that the University of Maryland has an abundance of historical material upon which to draw, and a special section devoted to this area seemed especially appropriate to the committee.

It is also noted that the Bylaws of the Alumni Association now permit membership to be drawn from the faculty of the School of Medicine. It seemed likely to the committee that if the faculty availed itself of this privilege, interest in the BULLETIN would be enhanced within the ranks of the faculty.

A new Editorial Board for the BULLETIN has been appointed in recent months. It is suggested that the new Editorial Board be apprised of this report and that their suggestions be solicited.

B U L L E T I N School of Medicine, University of Maryland

This quarterly publication, now in its 53rd year, is jointly owned and produced by the faculty of the School of Medicine, University of Maryland and by the Medical Alumni Association.

It is known as a multi-purpose institutional journal, carrying the following items:

a. Scientific articles of general or special medical interest
b. Book reviews
c. News and records of the School of Medicine
d. News of the Alumni Association and alumni
e. Obituaries
f. Special articles of para scientific and historic value

The four editions are comprised of approximately 64 pages each, with a press run of about 3,200 copies, the majority of which are subscribed for by the alumni of the School of Medicine. There are somewhat more than a hundred exchanges which are given to the Health Sciences Library with a cash value of about $900, a saving to the library costs. The journal is abstracted by *EXCERPTA MEDICA*, by *CHEMICAL ABSTRACTS* and is indexed in the *CUMULATED INDEX*. There is a small free circulation, as permitted by the postal authorities.

It costs about $11,000 a year to produce and distribute the BULLETIN. Of this, $3,400 is an appropriation from the Dean's office; the remainder from subscriptions or through appropriation by the Medical Alumni Association.

Dr. John A. Wagner serves as Editor. Mr. William J. Wiscott serves as Managing Editor. Mrs. Judith N. Manian has been recently appointed as Editorial Assistant. The BULLETIN is under the management of an Editorial Board, one half of which is appointed by the Medical Alumni Association and one half by the Dean of the School of Medicine. The President of the Medical Alumni Association and the Dean of the School of Medicine serve as ex officio members.

The BULLETIN serves the joint interest of the faculty, the School of Medicine, the University of Maryland Hospital and the Medical Alumni Association. It is sensitive to and obedient to the requirements of each which it seeks to satisfy in relationship to a total concept which includes a record of the passing events in the School of Medicine; of the achievement of its alumni and, hopefully, to carry a sample of the quality of research representative of that being undertaken in the School of Medicine. Plans for the BULLETIN include the possibility of a bi-monthly issue instead of quarterly and additional articles recording progress in medical science as it is currently being achieved at this School of Medicine. Now is the first time with adequate editorial assistance, the outlook for the future seems vastly improved.

The BULLETIN published 4 times a year is included in the alumni dues and is sent *gratis* to alumni during their first 2 years following graduation. The subscription prices and general policy as adopted by the Editorial Board are contained in the masthead.

President's Letter

Dear Fellow Alumni:

At the risk of being repetitious, I am going to take this opportunity to again discuss the project which is foremost in the minds of your Board of Directors, the restoration of Davidge Hall. This was the foremost topic of discussion at the October meeting of the Board of Directors of our Association.

It would further be our idea to establish this wonderful old building as a medical shrine. The Medical Alumni Association of the University of Maryland should bear the main financial burden of the restoration of Davidge Hall. The State, through the Medical School, should be responsible for the continued upkeep of the building and, of course, would be encouraged to use the building for classes, meetings and other educational functions.

I ask you all to keep Davidge Hall and its restoration in your thoughts and to contact me or any member of the Board of Directors if you have ideas concerning the accomplishment of this project.

Sincerely yours,

Lewis P. Gundry

Lewis P. Gundry, M.D.
President

ROSTER OF SENIOR ALUMNI

A distinguished group of physicians in practice more than 50 years comprises the Senior Alumni of the University of Maryland, the College of Physicians and Surgeons and Baltimore Medical College. The Alumni Association and the School of Medicine salute these honored physicians to acquaint younger alumni with their names and to acknowledge their service to medicine and to the community.

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One of the most outstanding alumni of the School of Medicine is **Dr. Rush B. Stevens** of the Class of 1908, who lives at 400 Hilgard Avenue in Los Angeles, California. Except for blindness Dr. Stevens is in good health. He has led an exemplary and most interesting life which he details to the Alumni Association and BULLETIN in this, his 86th year.

"Following my graduation I spent two years in the Mercy Hospital as a Resident Surgeon and also acted as an assistant teacher in Bacteriology and Pathology in the College. In 1910, I went West and started practice in Fillmore, Utah, the first Capital of Utah. I was one of the first doctors in that territory and the only doctor in the whole county, acting as pharmacist, dentist, and all around medical and surgical man for a population of over 10,000 people. This was before automobiles and I had to make my calls with teams and buggies to important cases sometimes 50 miles distance away. I remember buying my first car, a Dodge, in 1913 (for $800.00), which was the first car in that part of the country. I was appointed by the U. S. Government to look after the Pavant Tribe of the Ute Indians (over 500) during my stay in Fillmore. I later moved to Salt Lake City and practiced there until I retired in 1938. I have been Secretary of the State Medical Association and also on the first Medical Board of the State Industrial Commission for 3 years.

"I was Captain of the Army Reserve from 1916 to 1944. While in Fillmore, I married Anna Huntsman in 1911 and have one son, Weir C. Stevens, who is now Chief Surgeon of Kennecott Copper Co. in Arizona, and a Fellow of the American College of Surgeons, and one daughter, Beth Anne.

"I moved to Los Angeles in 1938 and built my home directly across the street from U.C.L.A., on a hill overlooking the Pacific Ocean.

"I would appreciate knowing the names and addresses of those still living who graduated in 1908."

CLASS OF 1920

Dr. J. Morris Reese was recently awarded a plaque in commemoration of his Presidency of the Medical and Chirurgical Faculty of Maryland.

CLASS OF 1928

Dr. Robert S. McCeney, was selected by the Prince George's County Medical Society as their candidate for the "Doctor of the Year."

CLASS OF 1929

Dr. Lewis M. Overton has been named Chief of the Department of Orthopaedic Surgery at the Veterans Administration Hospital in Albuquerque, New Mexico. Dr. Overton will also serve as Associate Professor of Orthopaedic Surgery at the University of New Mexico Medical School.

CLASS OF 1933

Dr. George S. Baker has been promoted to professor of clinical neurosurgery in the Mayo Graduate School of Medicine of the University of Minnesota.

CLASS OF 1935

Dr. Harry A. Teitelbaum has announced the relocation of his office for the practice of neurology and psychiatry, to 200 West Cold Spring Lane, Baltimore, Maryland 21210.

CLASS OF 1936

Dr. William C. Griefinger has been elected President of the medical staff of the Belleville Hospital in Newark, N. J.

CLASS OF 1937

Dr. Isadore Kaplan has been elected President of the American Association of Railway Surgeons. Dr. Kaplan serves as Director of Medical Services for the Chesapeake & Ohio Railway Company and the Baltimore & Ohio Railroad, which employ some 1800 doctors throughout the United States and Canada. The election was announced at the eightieth annual meeting of the association, which was held in Chicago.

A native of Baltimore, Dr. Kaplan joined the Baltimore & Ohio Railroad in 1946 as Assistant Medical Examiner, becoming Surgical Director in 1958. In 1967 he was named Director of Medical Services for both railway corporations.

A frequent contributor to medical and scientific journals, Dr. Kaplan is a member of the American Medical Association, the Baltimore City Medical Society and the Medical and Chirurgical Faculty of Maryland. He also serves as chairman of the Medical-Legal Committee, Medical Section, Association of American Railroads. During the war, Dr. Kaplan served with the 104th Medical Regiment of the 29th Division.

CLASS OF 1942

Dr. Joseph G. Bird has been named Director of Experimental Medicine of the Sterling-Winthrop Research Institute at Rensselaer, N. Y.

Dr. Albert L. Ingram, Jr. has been named Commissioner of Mental Health of the State of Delaware. A former Navy psychiatrist, Dr. Ingram has practiced psychiatry in Wilmington, Delaware and was Director of Psychiatry at the Delaware Hospital where he supervised the establishment of the first in-patient general hospital service for psychiatric patients in the state. In 1953 he served as the first President of the Delaware Psychiatric Society. Since 1963, he has been director of university health services at Pennsylvania State University.

CLASS OF 1944

Dr. Jose Alverez De Choudans has been elected President of the Puerto Rico Medical Society. Dr. Alverez was formally a resident in neurological surgery at the University of Maryland Hospital.

CLASS OF 1952

Dr. Robert N. Headley, a graduate of the University of Maryland, has been promoted to Associate Professor of Medicine at the Bowman Gray School of Medicine.

His promotion will become effective July 1.

Dr. Headley, a cardiologist, was appointed to the Bowman Gray faculty in 1963. He is Director of the Outpatient Department of the Medical Center and is Chairman of the Task Force on Heart Disease of the Carolina Regional Medical Program.

He received the B. S. degree (*cum laude*) from the University of Maryland in 1952 and the M.D. degree from the University of Maryland School of Medicine in 1965.

While in medical school he was a Mosby Scholar and the recipient of the Student Council Key. He was President of his junior and senior medical classes and was elected to *Alpha Omega Alpha,* national medical honor society.

Dr. Headley took postdoctoral training at the University of Virginia Hospital and North Carolina Baptist Hospital.

He is married to the former Wilhelmina Kerlee of Black Mountain, N. C. They have four children.

Dr. Irvin Hyatt has been named Chief of Medicine at the Baltimore County General Hospital.

Dr. Walter D. Gable has been named deputy chief medical examiner in the western division of the Office of the Chief Medical Examiner of Virginia.

CLASS OF 1953

Dr. Rafael Longo Cordero has been named President of the Medical Staff of the Presbyterian Hospital, San Juan, Puerto Rico Class of 1969.

CLASS OF 1955

Dr. Stanley P. Balcerzak, Jr., has been recently appointed associate professor of Medicine in the section on Hematology at Ohio State University. Dr. Balcerzak holds a certificate of the American Board of Internal Medicine specializing in hematology.

Following his internship he was appointed instructor in Medicine at the University of Chicago. For the next 2 years he served on the hematology service at the Walter Reed Medical Center and Walter Reed Army Institute of Research serving as hematologist. Prior to his appointment at Ohio State, he served as assistant professor of Medicine at the University of Pittsburgh. He is a member of *Phi Beta Kappa, Alpha Omega Alpha*, American Federation for Clinical Research, American Society of Hematology, American Board of Internal Medicine, and Fellow of the American College of Physicians.

Dr. Henry B. Higman has been named the first Chairman of the new Department of Neurology in the University of Pittsburgh's School of Medicine.

CLASS OF 1957

Dr. Peter P. Lynch of 4650 Walzem Road, San Antonio, Texas, writes that he will be delighted to see any former professors, classmates and friends at the "Hemisfair-1968", the multi-million dollar extravaganza taking place in San Antonio, Texas this summer. Dr. Lynch may be reached through the physicians exchange (226-3336) or at the above address.

CLASS OF 1958

Dr. Frank P. Greene has resigned from the U. S. Public Health Service and has joined the Red Bluff Medical Group at 737 Washington Street, Red Bluff, California 96080.

CLASS OF 1959

Dr. Lawrence D. Pinkner has moved his office to 200 W. Cold Spring Lane in Baltimore.

CLASS OF 1961

Dr. Milton H. Buschman is the recent author of an article entitled "Adjudications of Defendants on Tranquilizers Psychiatrically Evaluated Competent to Stand Trial" published in the March, 1968, issue of the *American Bar Association Journal.*

Dr. Nina Vann Jeanes of 1415 Third Street, Corpus Christi, Texas has recently completed her residency in Obstetrics and Gynecology and is now practicing this specialty in Corpus Christi, Texas.

CLASS OF 1962

Dr. Donald M. Barrick of 701 St. Paul Street, Baltimore, Maryland 21202, has been recently certified in general surgery of the American Board of Surgery.

Dr. William R. Law has been named director of the home health services program for Baltimore County.

CLASS OF 1963

Dr. Robert E. Dinker has been recently named to the faculty of the Bowman Gray School of Medicine as instructor in radiology.

Dr. Donald H. Gilden is currently serving in the Department of Neurology at the Walter Reed General Hospital, Washington, D. C.

Class of 1963 cont.

Dr. Thomas V. Inglesby of 130 W. 12th Street, New York, New York 10011 is completing his second year training in Internal Medicine at St. Vincent's Hospital and Medical Center, New York City. In 1968, Dr. Inglesby will begin a 2 year Fellowship in Cardiology at Emory University School of Medicine, Atlanta, Georgia.

CLASS OF 1965

Dr. David R. Harris has been appointed Chief Resident in Dermatology at Stanford University School of Medicine.

Dr. Philip Toskes, resident physician at the University of Maryland Hospital, has received a Mead Johnson scholarship from the American College of Physicians. The award, Dr. Toskes' second, is given annually to 10 residents in internal medicine who are receiving training within the U. S.

CLASS OF 1967

Dr. Robert O. France has announced the opening of his office in White Hall, Maryland.

ALUMNI NEWS REPORT

TO THE BULLETIN:

I would like to report the following: _____

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SUGGESTIONS FOR NEWS ITEMS

American Board Certification
Change of Address
Change of Office
Residency Appointment
Research Completed
News of Another Alumnus
Academic Appointment
Interesting Historic Photographs

Name _____

Address _____

_____

Class _____

Send to

Dr. John A. Wagner, Editor
Bulletin—School of Medicine
University of Maryland
31 S. Greene St.
Baltimore, Md. 21201

Deaths

CLASS OF 1902

Dr. Clarence E. Collins of Crisfield, Maryland, died August 12, 1968. Dr. Collins was 98.

Dr. Robert Oliver Lyell of 226 N. E. 20th Terrace, Miami, Florida, died February 29, 1968, at the age of 89.

CLASS OF 1904 BMC

Dr. Herman E. Hasseltine of Bristol, Vermont, died June 8, 1968.

CLASS OF 1904 P & S

Dr. David C. Mock of 215 Cajon Street, Redlands, California died January 30, 1968. Dr. Mock was 90.

CLASS OF 1905 BMC

Dr. S. Malcolm Magarian of 1930 St. John Road, Seal Beach, California, died February 20, 1968. Dr. Magarian was 87.

CLASS OF 1905 P & S

Dr. Benjamin B. Kasson of 1333 7th Street, San Diego, California died in March, 1968.

CLASS OF 1906 BMC

Dr. Edward F. Healy of 544 South Park Avenue, Buffalo, New York, died November 4, 1967, at the age of 85.

CLASS OF 1906 P&S

Dr. Walter D. Wise of 1120 St. Paul Street, Baltimore, Maryland, died July 22, 1968, at the age of 83.

CLASS OF 1907 P & S

Dr. Ernest M. Perry of Rocky Mount, North Carolina died in the early part of May, 1968. Dr. Perry was 86. A native of Franklin County, North Carolina and an alumnus of Wake Forest College, most of Dr. Perry's active professional career was spent in general practice at Rocky Mount, North Carolina.

CLASS OF 1908 BMC

Dr. Emmett A. Moore of Newark, Ohio, died May 12, 1968, at the age of 86.

CLASS OF 1908 P & S

Dr. Clyde W. Conn of Route 2, Uniontown, Pa., died recently.

CLASS OF 1909

Dr. Wilmer Marshall Priest of 55 W. 184th Street, New York City, died on August 3, 1967. Dr. Priest was 79.

CLASS OF 1910

Dr. H. M. Foster, 2220 Westchester Avenue, Catonsville, Maryland died November 23, 1967. Dr. Foster was 79.

CLASS OF 1911

Dr. John Hogan of Gibson Island, Maryland, died November 24, 1967. Dr. Hogan was 81.

CLASS OF 1912 P & S

Dr. S. J. Roberts of 1432 North 2nd Street, Harrisburg, Pa. died on August 20, 1965, at the Harrisburg Polyclinic Hospital.

CLASS OF 1912

Dr. Edwin V. Whitaker, Box 625, Baton Rouge, La., died January 15, 1968. Dr. Whitaker was 78.

CLASS OF 1913

Dr. James Sylvester Dixon of 908 Hooper Avenue, Baltimore, Maryland died December 25, 1967. Dr. Dixon was 80.

CLASS OF 1913 P&S

Dr. James Corbin Doughty of Onancock, Virginia, died June 15, 1968.

CLASS OF 1914

Dr. C. E. Dovell of 62 South Boxwood Street, Hampton, Virginia 23369, died on March 24, 1968.

CLASS OF 1916

Dr. Thomas Latham Bray of Plymouth, North Carolina, died August 10, 1967, at the age of 78.

CLASS OF 1916

Dr. A. B. Nevling of 426 Fifth Street, S.W., Rochester, Minnesota, died July 8, 1968.

CLASS OF 1918

Dr. Lang W. Anderson of Route 1, Williston, South Carolina, died suddenly on June 16, 1968, en route to Baltimore to celebrate his 50th anniversary.

CLASS OF 1920

Dr. Frederick A. Holden, formerly a counselor from Maryland of the Southern Medical Association and prominent ophthalmologist and otolaryngologist, died at the Union Memorial Hospital after a long illness. Dr. Holden was 71.

A native of Queenstown, Maryland, and an alumnus of St. John's College, Annapolis, Dr. Holder served as Associate Professor of Otolaryngology at the University of Maryland School from 1920-41. During World War II, he served in the U. S. Navy as Chief flight surgeon on the USS Tulagi with the rank of Commander. Dr. Holden retired from active practice in 1967. Dr. Holden was active in Rotary International and was a member of the Baltimore County Medical Association, the Medical and Chirurgical Faculty of Maryland and the American Medical Association.

Dr. William Leuders, Jr. of Route #1, Bel Air, Md., died May 11, 1968. Dr. Leuders was 70.

CLASS OF 1921

Dr. Felix S. Shubert of 3926 State Street, Erie, Pennsylvania died February 15, 1968.

Dr. John V. Szczerbicki, an East Baltimore practitioner for more than 40 years, died on August 12, 1968, at Church Home and Hospital after a long illness. Dr. Szczerbicki was 72.

Following his graduation from the School of Medicine, he served his medical and surgical internships at St. Joseph's Hospital, where he became a member of the senior staff.

CLASS OF 1922

Dr. Aaron H. Trynin, of 4711 12th Avenue, Brooklyn, New York, died August 22, 1967, at the age of 69.

CLASS OF 1923

Dr. Douglas A. Haddock of 11019 Balmwill, Norwalk, California died November 11, 1966.

CLASS OF 1926

Dr. Arthur N. Freuder of 365 Parkside Avenue, Brooklyn, New York, died November 4, 1967.

CLASS OF 1929

Dr. John Edward Murphy of 802 S. Valley Avenue, Oliphant, Pa. died March 1, 1968.

CLASS OF 1936

Dr. Theodore A. Schwartz, for many years Chief of the Department of Otolaryngology at the Mercy Hospital and an Associate Professor in the School of Medicine, died at his home in Baltimore on June 1, 1968, after a brief illness.

Formerly an associate of Dr. W. F. Zinn at the Mercy Hospital, Dr. Schwartz also was active at the Sinai Hospital where he had served as attending otolaryngologist since 1936. He also held privileges in his specialty at the Baltimore Eye, Ear and Throat Hospital, the Lutheran Hospital of Maryland, the Baltimore County General Hospital and the St. Mary's Hospital in Leonardtown, Maryland. He also served as a consultant at the Spring Grove State Hospital and at the Bon Secours Hospital.

From 1942 to 1946, he served as otolaryngologist at the University of Maryland's 42nd General Hospital with the rank of Lieut. Colonel. The alumni of the School of Medicine will long remember Dr. Schwartz as a competent surgeon, a fine gentleman, and an excellent teacher.

CLASS OF 1937

Dr. C. Parke Scarborough, Jr., died at his home on June 4, 1968, after a long illness. Dr. Scarborough was 55. Nationally known as a plastic surgeon, Dr. Scarborough served as Chief of this division in the Department of Surgery at the School of Medicine with a rank of Assistant Professor of Surgery. Dr. Scarborough also maintained offices in the Medical Arts Building in Baltimore.

A native of Delta, Pa. he heceived his bachelor's degree from Pennsylvania State College. His medical school career was distinguished by the fact that he was elected President of his class for all 4 years. His leadership and his personal qualities resulted in his being nominated President of the Medical Alumni Association for the year 1966, serving in this capacity with distinction.

He was an active practitioner devoted to the precise aspects of his specialty, serving not only on the Instructional staff of his Alma Mater but also was active as a staff member of nearly all of the prominent Baltimore hospitals. Dr. Scarborough served as a member of the Board of Trustees of the Maryland Medical Service (Blue Shield) and for a time served as Chief Consultant to the Maryland State Industrial Commission.

During World War II, he was active at the Plastic Surgeon Center at Valley Forge, Pa. becoming the Chief of Surgery at this faculty in 1946. Following the war, he served as consultant in plastic surgery, Veterans Administration.

For many years, he served as secretary and was a former President of the Lister Society of Baltimore. He was a member of the Baltimore City Medical Society, Medical and Chirurgical Faculty of Md., American Society of Plastic and Reconstructive Surgery, and a diplomate of the American Board of Plastic Surgery.

A friendly man of wide interests, not only an eminent scientist and a devoted physician, Dr. Scarborough had numerous other interests which included Sons of American Revolution, Sons of Colonial Wars, St. Andrew's Society, Maryland Historical Society, the Baltimore Symphony Orchestra Association, the Baltimore Country Club, and the Rehoboth Beach Country Club.

CLASS OF 1938

Dr. John P. Smith, age 58, died at his home 27 September, 1968 after a brief illness.

A native of Baltimore, Dr. Smith received his pre-medical education at the Johns Hopkins University. Following his gradution, he served for 5 years in World War II as an Army medical officer in the European Theatre. Following the war, he served his residency at the Maryland General Hospital, establishing his practice in 1947.

CLASS OF 1943

From a contemporary in the subsequent "war" class of December, 1943, comes a brief and poignant comment on the recent death of **John Epperson.** Crosby states that, "a notation of two sentences did not do justice to the expansive demeanor of this true son of the hills of West Virginia."

"My first real encounter with him was at Baltimore City Hospitals when I was the first neurosurgical resident. After I had been there for a period of several weeks and had gotten to know John who was then serving as a resident in obstetrics, I was astounded one evening at suppertime as John walked into the large House Officers' Dining Room, pointed over to the corner table where I sat with the general surgical house officers, and in his very loud voice informed all present, 'There he is, the neurosurgeon, the only man who makes rounds in the morgue.' Needless to say, this stimulated laughter that I think had never been heard before and I am sure was never heard again at City Hospitals.

"When one got to know John better, he was amazed at the sense of humor that John brought to most every situation. He had sustained a fracture of the elbow at an earlier age and, as a result, had some damage to his ulnar nerve. On three separate occasions neurosurgeons noted the ulnar

palsy position of his hand with the fourth and fifth fingers partially flexed on the palm and suggested to John that they could repair his ulnar nerve so that these fingers would work again. John would then lead them on to the point of getting him about 24 hours prior to admission to the hospital for surgery on his ulnar nerve and then he would sit down with the surgeon and say, 'Doctor, someone has been telling me about causalgia. Would you explain this to me and how this will affect my hand and arm after you operate on my ulnar nerve?' This invariably ended the entire adventure.

"I was somewhat saddened to read of the passing of this pre-World War II, slightly wild, but ever fascinating soul."

R. M. N. Crosby, M.D.
Class of 1943

CLASS OF 1945

Dr. Robert DeWitt Peck of Montgomery, West Virginia, died suddenly on September 8th, apparently having suffered a heart attack.

A life long resident of Fayette County, West Virginia, Dr. Peck was a member of the Laird Memorial Hospital.

An alumnus of the Montgomery High School and West Virginia University, he also attended Hampden-Sydney University prior to his enrollment in the School of Medicine.

Dr. Peck served in the Medical Corps in World War II, and was also active in Rotary International and in the Montgomery Chamber of Commerce. He was also past President of West Virginia County Medical Association.

CLASS OF 1956

Dr. Thomas A. Love, III, son of William S. Love, Jr., retired cardiologist and former faculty member, was killed in an automobile accident near Thurmont, Maryland, on April 3, 1968. Dr. Love was 35.

Dr. Love who was engaged in general practice in Thurmont was a graduate of Hereford High School and the University of Maryland. Following his graduation, he served an internship at Mercy Hospital. He was the grandson of the late Dr. William S. Love who died in 1940. Dr. Love was a member of the Chirurgical Faculty of Maryland and the Frederick Medical Society.

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BULLETIN

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